Exhibit 1

CTEH CENTER FOR TOXICOLOGY AND ENVIRONMENTAL HEALTH, LLC

COMMENTS ON THE HEALTH RISK BASIS FOR U.S. ENVIRONMENTAL PROTECTION AGENCY'S PROPOSED REGULATION OF CERTAIN USES OF METHYLENE CHLORIDE AND N-METHYLPYRROLIDONE UNDER TSCA 6(a)

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Executive Summary

The United States Environmental Protection Agency (EPA) intends to prohibit the manufacture, processing, and distribution in commerce of MeCl2 (MeCl2) for all consumer and for most types of commercial paint and coating removal uses. Likewise, EPA intends to either prohibit the manufacture, processing, and distribution in commerce of n-methylpyrrolidone (NMP) for all consumer and commercial paint and coating removal uses, or propose a reformulation, use of personal protective equipment (PPE), and labeling change approach to user risk reduction. These actions are based on EPA's Toxic Substances Control Act (TSCA) Work Plan risk assessments for each chemical that identify what the Agency considers to be unreasonable health risk to some or all users of paint stripping products containing MeCl2 or NMP.

Center for Toxicology and Environmental Health (CTEH) evaluated EPA's technical approach and health-related bases for these decisions. CTEH has determined the Agency has not used methods that reflect the best available science. As discussed below, CTEH has identified several significant shortfalls in the Agency's risk evaluation for potential acute risks to consumers who may use retail paint stripper products, such as those made and sold by WM Barr. The concerns CTEH has identified appear in both the hazard and exposure portions of the Agency's risk assessments for MeCl2 and NMP. In particular, CTEH identified the following overarching concerns regarding the EPA risk assessments for MeCl2 and NMP.

1. EPA's consumer exposure modeling methods are overly conservative and reflect assumptions that are unsupportable and do not accurately reflect consumer behaviors during use. EPA employed a Multi-Chamber Concentration and Exposure Model (MCCEM) to estimate consumer exposures during residential use scenarios. For reasons discussed in Section 2.0 below, CTEH concludes that the Agency's consumer exposure model greatly exaggerates the likely exposures that consumers who use paint strippers in residential use setting actually experience. Consequently, the model does not represent a reasonable worst case representation of such uses, and is inconsistent with the best scientific methods, and EPA's scientific standards. In 2016, after EPA had invited peer review of its MeCl2 and NMP risk assessments, the Agency undertook supplemental exposure analyses using enhanced consumer exposure modeling scenarios. The resulting supplemental analyses of consumer exposures for MeCl2 and NMP paint strippers have not been subject to peer review. EPA explained that the supplemental exposure scenarios modeled in the 2016 updates were designed to reflect additional use scenarios "with extended application times, increased product use and altered user behaviors." However, both the original scenarios EPA modeled, and the expanded scenarios modeled in 2016, do not reflect reasonable assumptions about the predominant location in which paint stripping work is typically undertaken by residential users (i.e., outdoors). Additionally, several scenarios modeled do not reflect that the product user leaves the location where a paint stripper is used both during breaks or immediately after the work is done; this is inconsistent with consumer behavior as reported in survey data that EPA has reviewed. In both its risk assessment and the enhanced scenarios, EPA modeled consumer exposures during bathtub stripping, even though EPA's risk assessment specifically states that EPA assumes professionals perform bath tub stripping. The scenarios EPA has modeled all assume consumers will repeatedly reapply even the most effective paint stripper products (MeCl2-based paint strippers), even though information provided to EPA documents that removal of multiple layers of a wide variety of coatings is achieved rapidly

with retail MeCl2 paint strippers, and without the need to reapply the product. These and other EPA assumptions result in a model that overstates likely consumer exposures during residential uses

2. EPA relied upon candidate Points of Departure (PODs) from previously developed toxicity assessments that do not use best scientific practices and methodologies currently available at the time of the MeCl2 assessment. Central nervous system (CNS) effects are the acute adverse health impacts observed from sufficiently high MeCl2 exposures. Effects from repeated, daily exposures (not relevant to potential acute exposures from consumer uses) are different from acute exposures and result from repeated contact for MeCl2 with specific organ systems over longer periods of time. EPA did not derive its own Point of Departure (POD, a chemical exposure level that represents an approximate threshold of adverse effect), but rather considered four different PODs taken from previously-performed assessments of acute human MeCl2 toxicity. Those PODs were comprised of a National Research Council Spacecraft Maximum Allowable Concentration (SMAC), a California Office of Environmental Health Hazard Assessment (OEHHA) acute Reference Exposure Level, and two National Academies of Science Acute Exposure Guideline Levels (AEGLs). For purposes of the 2014 Risk Assessment and its 2016 Supplement, the Agency ultimately relied on the SMAC-based POD. A POD that is protective of the general public, taking into account susceptible subpopulations, would be preferable to using a SMAC-based POD.

The AEGL values represent a more appropriate set of PODs, as they were derived using a Physiologically Based Pharmacokinetic (PBPK) model to simulate inhaled air, brain, blood, and metabolized levels of MeCl2 for specific potential exposures. The ability to simulate these levels is one of the advantages of using well-developed PBPK models in human health risk assessment. EPA and NAS have previously recognized the use of PBPK models for this purpose. In fact, these types of models have been used by the Agency to derive chronic human toxicity values for multiple chemicals in the past, including MeCl2. EPA did not explain why its selection of the SMAC-based POD is preferable to the AEGL-based PODs, particularly with the better exposure extrapolation and POD determination capabilities afforded by using a PBPK model. EPA should have utilized a PBPK model for MeCl2 that it used for the 2011 IRIS assessment. That model was calibrated based on human population and individual data, accounting for both scientific uncertainty and experimental variability inherent in the individual model parameters. That model also used probabilistic parameter distributions to simulate a POD, which are preferable to relying on a single exposure level from a single study, as was done by EPA for applying the SMAC-based POD. Peer-reviewed publications and an EPA white paper all discuss the advantages of using probabilistic methods in risk assessment.

3. EPA's POD and benchmark Margin of Exposure (MOE) selection introduced unnecessary scientific uncertainty into the estimation of acute health risks. EPA has utilized benchmark Margins of Exposure (MOEs, values that account for POD uncertainty) that introduce additional and unnecessary uncertainty into the calculation of actual MOEs (ratios of POD to likely exposure level). EPA's use of a benchmark MOE of 10 associated with the Agency's preferred SMAC-based POD would have been obviated if one of the PBPK model-based AEGL values (better science), or a POD developed using the 2011 IRIS PBPK model (best available science), had been used. As it is, EPA's use of SMAC-based MOEs creates an unnecessary level of conservatism that cascades throughout the risk assessment.

The difference in uncertainty factors that define the benchmark MOEs for the four different EPA PODs is quite striking, and illustrates the need to apply the best science to POD/benchmark MOE selection. The use of a probabilistic PBPK model to extrapolate an observed effect (COHb production and CNS depression) from a study to various exposure durations in humans, and the inclusion of the biology of MeCl2 metabolism and elimination into the exposure calculation, results in a POD, its benchmark MOE, and low-risk exposure estimates that are less biased by uncertainty. The EPA-modified PBPK model used in the 2011 IRIS assessment for MeCl2 should have been used by EPA to bring its MeCl2 risk assessment in line with its own technical standards.

4. Use of the best available PBPK model to derive a POD and associated benchmark MOE would result in lower consumer risk estimates than those proposed by EPA. The most-advanced, vetted, and available PBPK model for MeCl2 should be used to simulate potential acute inhalation exposures and derive a POD, taking into account variability in population physiology, metabolic capacity, ethnic variations of metabolism, variations in COHb production, and exposure duration-specific effects on internal doses of blood MeCl2 and COHb levels. EPA's failure to use a PBPK-based MOEs enabled the Agency to support its conclusion regarding unreasonable risk for consumer use of MeCl2 for paint stripping purposes.

5. Use of alternative compounds instead of MeCl2 will not reduce acute health risks to consumer users based on CTEH's analysis performed using EPA methodology. . EPA published an assessment of toxicity and exposure potentials for mixtures containing various combinations of 14 individual compounds selected as key ingredients for alternative products. Several of the 14 key ingredients identified by EPA are highly flammable materials, resulting in formulations presenting a physical health hazard that is not present for users of MeCl2 paint strippers. Although many MeCl2-containing products also contain some flammable ingredients, replacing MeCl2 in such formulations with a higher concentrations of the same or an additional flammable compound will results in an alternative paint stripper that presents a greater risk of thermal burn and smoke inhalation injuries to residential users.

CTEH reviewed the toxicology data for the 14 key alternative compounds, as well as for mechanical sanding particulates. CTEH also conducted consumer use simulation modeling for 8 of the alternative compounds to provide exposure estimates for performance-equivalent uses of the alternatives that are directly comparable to various scenarios used by EPA to determine risks to consumers using MeCl2. (Such modeling could not be conducted for compounds that did not have adequate toxicity data for use in acute risk assessment or which did not present an inhalation hazard.) CTEH used WM Barr (2017) performance equivalence data to determine reasonable estimates of the amount of an alternative product that must be applied to achieve performance-equivalence with MeCl2 strippers and the necessary residence (wait, or "dwell") times for each alternative compound. CTEH derived benchmark MOEs to account for data uncertainty and calculated risk values as MOEs for the alternative compounds, as EPA did for MeCl2. CTEH's risk calculations, using EPA's methodology, showed that only one class of compounds (dibasic esters) resulted in consumer health risks that would not be deemed unreasonable using EPA's criteria. However, performance data for such paint stripper products shows them to be ineffective coating removers.

6. EPA's 2015 NMP risk assessment does not indicate unreasonable risk of potential short-term health effects to consumers, but does introduce overly conservative uncertainty into its risk calculations. For acute consumer uses, EPA's approach to estimating inhalation exposure for NMP was generally the same as was taken for MeCl2. However, the NMP risk assessment utilized a PBPK model for NMP in rodents and pregnant/child-bearing aged women to simulate the critical developmental toxicity study and extrapolate internal NMP doses across species and to specific consumer NMP use scenarios. The human PBPK model was used to determine aggregate dermal and inhalation exposures that would result in the equivalent blood levels believed to not convey a risk for developmental effects in humans. This process represents the use of best science to determine risk. Nevertheless, EPA did not adequately justify its use of an additional uncertainty factor of 3 (to account for human inter-individual variability in NMP pharmacokinetics) in its risk calculations. Use of the PBPK model, whose parameters are average values based on a range of data, reflects dynamic biological processes involved with the changing body of pregnant women and fetuses in general, resulting in an internal NMP dose likely to occur in pregnant women, a sensitive subpopulation. Removing the unnecessary uncertainty factor from the NMP risk calculations would not only indicate a higher margin of safety for single or short-term consumer uses of NMP as a paint stripper, but would also change EPA's determination of unreasonable risk involved with some occupational uses as well.

CTEH evaluated EPA's technical approach and health-based bases of these decisions, and found several significant shortfalls in the Agency's risk evaluation for potential acute risks to consumers who may use retail-size paint stripper products, such as those made and sold by WM Barr. EPA's TSCA risk assessment for MeCl2 did not leverage the best available science, and offered a conclusion of unreasonable risk to consumers based on questionable methodology to determine a POD and benchmark MOE. Although, for NMP, EPA did use an available PBPK model to derive PODs, benchmark MOEs, and actual MOEs, the Agency's unnecessary use of an additional uncertainty factor enabled EPA to conclude that consumer residential uses of NMP paint strippers may experience an unreasonable risk.

To reflect the best scientific methods and the information reasonably available to the Agency, EPA must reevaluate its assessments of consumer risks to the retail-size containers of MeCl2 and NMP paint stripper products. First, EPA must revise its modeling to more realistically and reasonably reflect consumer use scenarios -- including the fact that paint strippers are predominantly used outdoors and the lack of need for repeat applications. Second, EPA must employ the latest probabilistic PBPK model for predicting hazards of MeCl2 exposures, using the methods the Agency used for its 2011 IRIS assessment of MeCl2 and the techniques it used for its NMP assessment. Finally, EPA must chose uncertainty factors benchmark MOEs that are appropriate to the use of PBPK modeling, rather than the benchmark MOEs EPA selected for MeCl2 and for NMP that are not necessary and reflect overly conservative values and overstate consumer risks.

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1.0 Introduction

Methylene chloride (MeCl2) and n-methylpyrrolidone (NMP) are organic compounds used in formulations of paint and coating removal products sold in the U.S. Their inclusion in these products is driven by their ability to penetrate coatings and disrupt the coating's adhesion to the surface of the painted/coated object, typically wood, metal, or masonry. On January 19, 2017, the Unites States Environmental Protection Agency (EPA) (2017) issued a notice that it intends to "*…prohibit the manufacture (including import), processing, and distribution in commerce of methylene chloride for all consumer and for most types of commercial paint and coating removal uses*." For NMP, the notice stated that EPA intends to either "*…prohibit the manufacture (including import), processing, and distribution in commerce of NMP for all consumer and commercial paint and coating removal…*", or "…[propose] *a reformulation, PPE, and labeling approach. This would require product reformulation to limit the concentration of NMP in paint and coating removal products; testing of product formulations to identify specialized gloves that provide protection; relabeling of products to provide additional information to consumers; an occupational dermal and respiratory protection program for commercial use of NMP in paint and coating removal, downstream notification when distributing NMP for other uses, and limited recordkeeping.*" These proposed regulatory actions are based on EPA's opinion (USEPA, 2017) that the present availability of either compound in products used for paint or coating removal "will present an unreasonable risk of injury to health or the environment." (TSCA 15 U.S.C. §2605(a)).

WM Barr, Inc., is a U.S. corporation and a producer of consumer products including paint and coating remover products that contain MeCl2 or NMP and are sold in retail stores in the U.S. WM Barr's products that contain these two compounds are sold as either 1- quart or 1-gallon cans of liquid, or as 18-ounce aerosol spray cans. Due to the product container size and sales and retail marketing of its products, most uses of WM Barr's MeCl2 and NMP-containing products are by consumers involved with do-it-yourself type refinishing projects, or contractors performing paint or coating stripping tasks similar to those of consumers.

In the present report, Michael H. Lumpkin, Ph.D, DABT, a senior toxicologist with the Center for Toxicology and Environmental Health, LLC (CTEH) provides a critical evaluation of the technical approach used by EPA to make the determination of unreasonable health risks for uses of MeCl2 relevant to WM Barr products: single or short-term consumer exposures associated with single-project paint or coating removal at home. Dr. Lumpkin's curriculum vitae is provided in Appendix A. Occupational exposures and risks were not evaluated by CTEH as these uses would likely involve purchase of MeCl2-containing products in greater volumes than are available for WM Barr products. CTEH also provides comments on specific approaches used, and concludes EPA did not base risk calculations for MeCl2 on the best available science, which EPA established as an important goal to support science-based assessments of health risk (USEPA, 2014a).

CTEH is an environmental and human health consultancy specializing in health risk assessment and regulatory compliance, as well as responding to hazardous materials emergencies and chemical releases. Toxicologists, industrial hygienists, safety professionals, and environmental scientists at CTEH have been assessing worker and consumer exposure and public health issues since 1997. CTEH has performed

thousands of toxicology, exposure, and risk assessments for clients in the occupational health, consumer product, pharmaceutical, energy, and transportation industries, as well as state and federal agencies.

1.1 Overview of EPA's health risk assessment technical bases supporting the proposed regulation

The proposed regulation for MeCl2 and NMP states that the regulated uses of these chemicals in occupational and consumer settings may result in unreasonable risk (criteria defined below) to human health, based on analysis presented in the 2014 and 2015 TSCA Work Plan Risk Assessments for MeCl2 and NMP, respectively (USEPA, 2014b; USEPA, 2015b). Briefly, both risk assessments described typical commercial and consumer uses of the respective chemicals, provided descriptions and results of survey-based or computer-simulated exposures during commercial and consumer uses, reviewed the toxicology data for acute and chronic effects, and provided risk characterization calculations for short-term and chronic commercial and consumer uses and chronic commercial uses of MeCl2 or NMP. The following discussion details each of these steps in the two risk assessments for MeCl2 and NMP uses relevant to WM Barr products: consumer exposures from single project applications of paint or coating strippers.

The EPA (USEPA, 2014b; USEPA, 2015b) summarized data that informed the exposure factors likely to impact single or short-term consumer MeCl2 and NMP uses. From these data, as well as data from tests of MeCl2 product use in controlled laboratory environments, EPA developed seven single exposure scenarios involving brush-on or spray-on applications to furniture or a bath tub in a hypothetical in-home workshop or bathroom, respectively. EPA did not consider some important exposure scenarios: paint stripping outdoors or in a garage with the main door opened. The EPA Multi-Chamber Concentration and Exposure Model (MCCEM) software was used to simulate the exposure scenarios. The MCCEM utilizes mathematical relationships of building ventilation, user activity patterns, and chemical evaporation behaviors to calculate air concentrations in one or two zones within a building, as well as inhalation exposure levels for a user of a chemical product (USEPA, 1991). Using this model, the EPA calculated average exposures to a hypothetical MeCl2 user and bystander. These averages were compared to the toxicological data to provide an estimate of potential acute, non-cancer risk of adverse effects.

In recent years, W.M. Barr introduced new labeling for its retail paint stripper products that specifically warns consumers against using Barrr's products to strip bathtub coatings. As noted above, EPA stated that bathtub stripping is performed by professional, rather than consumers. Thus, CTEH considered the exposure scenario for bathtub refinishing reported in the 2014 risk assessment and 2016 supplemental exposure assessment to be irrelevant to likely consumer uses of WM Barr products.

The toxicological data defining the critical health effects and relevant exposure levels for single MeCl2 exposures were summarized by EPA. From this summary, EPA opined that the critical adverse health effects from single or short-term (hours) exposures directly (by virtue of central nervous system [CNS] depression) or indirectly (CNS oxygen deprivation due to carboxyhemoglobin formation in the blood) resulted in neurological impairment. Four toxicological Points of Departure (PODs), discussed below in Comment #1, were identified as candidate values for use in the EPA risk assessment. These four candidate PODs came from four previously developed assessments of the MeCl2 data. A POD is a chemical exposure

level that represents an approximate threshold of adverse effects as identified in epidemiology, lab animal, or human volunteer studies. Typically, the POD is the observed or estimated No Observed Adverse Effects Level (NOAEL), a dose that does not produce an adverse effect in a particular study. In the absence of NOAEL information, a Lowest Observed Adverse Effects Level (LOAEL) may be used (COC, 2014; USEPA, 2000).

For acute, non-cancer adverse health effects, EPA (2014b) calculated Margins of Exposure (MOEs) for each of the seven modeled exposure scenarios that it used to determine whether MeCl2 use by consumers represents an unreasonable health risk. A MOE is a ratio of a POD and a likely exposure level. The difference in magnitude between the POD and likely exposure is a semi-quantitative indication of risk. The lower the likely exposure level is in comparison to the POD (resulting in a larger MOE), the lesser the expected risk of the occurrence of adverse effects (USEPA, 2013).

PODs may be used in risk assessments as the starting point for extrapolating the estimated threshold for toxic hazard of a chemical from the study in which it was observed to a human population in general. The uncertainties in differences in the toxic threshold observed in a study of a limited number of laboratory animals or humans and potentially experienced by the human population as a whole may not be well understood. Therefore, numerical Uncertainty Factors (UFs) derived to account for potential or known differences may be applied to the POD. Uncertainty factors have been widely used in chemical risk assessment for decades (USEPA, 2014b) to "err on the side of caution." One way to do this is to aggregate all of the uncertainty factors (UFs) into a single number and divide the POD by that number. The resulting value is an exposure level which humans (including sensitive individuals) may experience that will not likely cause an adverse health effect. A similar use of PODs is to define an associate benchmark MOE. The benchmark MOE is a numerical value equal to the aggregate UF. The MOE (the ratio of the POD to the actual or likely exposure level) is compared to the benchmark MOE. If the MOE is higher than the benchmark MOE, then adverse effects resulting from the actual or likely exposure are not likely to occur. If the MOE is lower than the benchmark MOE, then an adverse effect may potentially (but not necessarily) occur. EPA defines this as an unreasonable risk. EPA used the MOE/benchmark MOE analysis in the MeCl2 and NMP risk assessments for short-term consumer risks.

Using the MOE methodology and MCCEM-simulated exposure estimates, EPA presented MOEs for a hypothetical user and bystander that indicated unreasonable risk (i.e., a MOE less than its associated benchmark MOE) for the seven single consumer MeCl2 use scenarios (USEPA, 2014d). It is upon these values (and similar analyses of single/short-term and chronic occupational exposures not relevant to WM Barr products) that EPA based its conclusion that consumer uses of MeCl2 result in an unreasonable health risk that necessitates regulatory action. EPA also produced a supplement to its 2014 risk assessment that addressed additional exposure scenarios for consumer use of MeCl2 products for paint and coating removal (USEPA, 2016b). This analysis used the same methodology that was used in the 2014 assessment, and concluded that the additional exposure scenarios examined also imparted "unreasonable risk" to consumer users.

In 2015, EPA issued the TSCA Work Plan Risk Assessment for NMP. The technical approach used to characterize risk to occupational and consumer users of NMP was similar to that taken for MeCl2, except

that rather than relying on previously developed toxicity assessments to identify one or more PODs, a physiologically-based pharmacokinetic (PBPK) model for NMP in rodents and humans was used to develop a POD for developmental effects on the fetus of pregnant women (USEPA, 2016b). A PBPK model is a computer simulation of chemical uptake, distribution, metabolism, and elimination in the body, utilizing mathematical representations of physiological, biological and biochemical processes to predict tissue levels of a chemical following a user-defined exposure. The advantages of using PBPK models to assess human health risks are presently discussed in Sections 2.2 and 5.1.

EPA used the same MCCEM simulation methodology used in the MeCl2 risk assessment (2014d) to estimate airborne NMP levels from which consumers would likely receive inhalation exposures. Dermal exposures, which included continuous exposures to at least half of the skin area of hands and fingers for up to four hours, and inhalation exposure were simulated using a PBPK model to provide internal dose estimates for each exposure scenario. MOEs were calculated based on these internal doses. The internal dose POD was also estimated using the PBPK model. The EPA did not identify MOEs lower than the selected benchmark MOE, and thus did not find an unreasonable risk to human health from potential single or short-term consumer exposures to NMP. However, EPA stated that "Based on a qualitative analysis of the outcomes it is possible that exposures of 4 or more hours could present risks comparable to those associated with short-term worker exposure scenarios." This "qualitative analysis" is simply an observation that longer exposures may result in increased risk. EPA provided no data suggesting that consumers would maintain or allow NMP to remain continuously on their hands for longer than four hours. There were no other statements in the NMP (2015b) risk assessment suggesting that NMP use by consumers would result in an unreasonable risk of injury to health.

In order to provide a further basis for findings of unreasonable risk to consumers using paint strippers, EPA also produced supplements to the 2014 MeCl2 and 2015 NMP risk assessments that addressed additional exposure scenarios for consumer use of products containing each chemical for paint and coating removal (USEPA, 2016b; USEPA, 2016c). These supplemental analyses intentionally involved larger project scenarios that would require greater use of paint stripping products over a longer time period. For example, it examines consumer users of paint strippers applying the paint stripper a third time to the entire surface of a bathtub in a small room. This is not necessary for MeCl2 products, and it contradicts the Agency's own conclusion that bathtub stripping is performed by professionals. (EPA 2014 MeCl2 Risk Assessment, Appendix H found at page 224 of 279.) The supplemental analyses were not subject to scientific peer review, as the 2014 and 2015 risk assessments were, nor did EPA provide maximum average concentrations for time intervals other than one hour, as was done in the 2014 and 2015 risk assessments. CTEH cannot conclude that the 2016 supplements reflects the best available science because the supplements appear to have been designed specifically to reflect unlikely and extreme exposure scenarios, rather than to generate approximations of reasonable worst case consumer exposure scenarios. Moreover, the 2016 supplements have not been peer reviewed.

Finally, EPA developed an assessment of chemical alternatives for use in place of MeCl2 for paint stripping. A search of alternative (non-MeCl2) products for sale in the U.S. identified a list of 76 candidate ingredients, which EPA pared down to a list of 14 key ingredients. The 14 ingredients were briefly reviewed for toxicological effects, with NOAELs identified. Relative inhalation and dermal exposure potential was

characterized by physical characteristics of each ingredient, such as vapor pressure, molecular weight, and the ingredients' tendency to partition from water to non-polar solvents (an indication of dermal absorption potential). The assessment did not quantitatively assess health risks from use of any of the ingredients, but rather compared the general toxicity and potential for evaporation to air and inhalation or skin absorption of each ingredient to that of MeCl2.

1.2 CTEH approach to evaluating the technical basis of the proposed regulation

CTEH reviewed the technical basis for EPA's health-related justification for the proposed regulation of MeCl2 and NMP and evaluated the scientific merit of both the methodology employed and resulting scientific conclusions to determine if the regulation is justified from a health risk reduction standpoint. CTEH evaluated whether the best available scientific practices for toxicological, exposure, and risk assessment were brought to bear in assessing risk from potential single or short-term consumer exposures to these two chemicals during paint or coating removal activities. Such adherence to best practices comports with the Toxic Substances Control Act (TSCA) mandate and EPA's stated objective to leverage best scientific practices to underpin the Agency's regulatory decisions. In its 2014 Strategic Plan document (USEPA, 2014a), EPA stated, "*We will continue to affirm the core values of science, transparency, and the rule of law in addressing our environmental challenges. Our work will be guided by the best possible data and research and a commitment to transparency and accountability.*" In discussing methodological changes to its approach to toxicity assessments developed for the Integrated Risk Information System (IRIS) program (USEPA, 2015a), EPA said, "*Ultimately, these changes will help EPA meet the goal of using the best available science to produce high quality scientific IRIS assessments in a timely and transparent manner.*" Finally, TSCA, as amended by the Frank R. Lautenberg Chemical Safety for the 21st Century Act (2016, Lautenberg Act), states that "*the [EPA] Administrator shall use scientific information, technical procedures, measures, methods, protocols, methodologies, or models, employed in a manner consistent with the best available science...*" Given these explicit standards, the present proposed regulation should be underpinned by data, tools, and analyses of the highest scientific caliber.

EPA relied primarily on the conclusions of the 2014 TSCA Work Plan Risk Assessment for Methylene Chloride (USEPA, 2014d), the 2015 TSCA Work Plan Risk Assessment for NMP (USEPA, 2015b), and the 2016 supplemental consumer exposure assessments for MeCl2 and NMP(USEPA, 2016a; USEPA, 2016b) to determine that consumer uses of MeCl2 and NMP products resulted in unreasonable risks. Thus, CTEH reviewed the parts of those risk assessments dealing with consumer exposures and evaluated the scientific rigor of the work.

CTEH reviewed EPA's procedures for estimating representative potential acute exposures for MeCl2 and NMP use for finish stripping by consumers. This evaluation included review of EPA's selection of representative product use scenarios, exposure factors related to these activities being carried out in a hypothetical home workshop environment, laboratory data used to define MeCl2 volatilization into the air, use of the MCCEM, and use of a PBPK model to simulate user dosimetry from the MCCEM-simulated exposures (NMP only).

EPA has expressed concerns about the use of MeCl2 to strip bathtub glazing in poorly ventilated bathrooms as it has been the cause of certain fatalities associated with CNS depression, loss of consciousness, and asphyxiation over the years (Chester et al., 2012). As discussed in a report submitted with Barr's comments, as prepared by Steve Hall of Applied Safety and Ergonomics, these fatalities did not involve consumer users; rather, they occurred when professional contractors were working in bathrooms with no or very low ventilation. Although EPA modeled user exposure for a bathtub refinishing exposure scenario, the estimated exposure values were lower than those recorded or estimated for the occupational or worker bathtub refinishing values, that led to reports of fatalities (which ranged from approximately 73,000 mg/m^3 (NAS, 2009) to over 300,000 mg/m^3 (Chester et al., 2012). EPA's modeling of a similar bathtub stripping activity under extreme conditions has resulted in estimated tub workspace air levels of over 16,000 mg/m^3 (USEPA, 2016b). For the reasons discussed previously, CTEH did not evaluate bathtub stripping under these conditions as a likely consumer scenario, as product labeling clearly informs users of the need for adequate ventilation. CTEH understands there is a petition pending before the CPSC that would codify the labeling directive on retail-size consumer use MeCl2 paint strippers specifically warning the products are not to be used for bathtub stripping.

CTEH also reviewed the toxicology summaries for acute, non-cancer effects provided in the MeCl2 and NMP risk assessments. For MeCl2, CTEH evaluated the EPA's summaries of the four toxicity assessments on which candidate PODs were based. Further, the basis of the acute consumer benchmark MOEs were reviewed. The benchmark MOEs and calculated MOEs were also reviewed. These values were ultimately relied upon the EPA to justify its conclusion that MeCl2 use by consumers results in unreasonable risk, and that similar use of NMP results in the same.

Considering the relative risks of single or short-term exposures resulting from consumers uses of alternative compounds identified by EPA, CTEH reviewed EPA's Analysis Report of Chemical Alternatives (USEPA, 2016a), WM Barr's study on paint stripping performance of alternative compounds (WM Barr, 2017), and other documents qualitatively describing the stripping performance and physical/chemical characteristics of alternative compounds compared to MeCl2 (Acywaye et al., 2015; Boast, 1992; Puglionesi et al., 1995; Wilson and Masatsugu, 2014). These documents were reviewed to determine the equivalent amount of compound to be utilized and the time required for an alternative to remain on the coating surface (residence time) in order to provide a stripping performance approximately equivalent to that of MeCl2. This relationship is defined as performance equivalence. The WM Barr (2015) study was judged to be the best source of data for performance equivalence because MeCl2, individual alternative compounds, and stripping products containing MeCl2 or alternative compounds were tested in an identical, controlled fashion on surfaces coated with three distinctly different paint types (alkyd, epoxy, and automotive primer/paint/clear coat applications).

CTEH performed MCCEM simulations of brush-on and spray-on applications of 8 of the 14 compounds/compound classes listed by USEPA (2016a) as suitable alternatives by EPA, using the same use scenario and building size/ventilation parameters used by USEPA (2014d) for MeCl2. Four of the 14 compounds or compound classes were not assessed for performance-equivalent inhalation exposures because of their non-volatilization properties (caustic hydroxides), lack of adequate acute inhalation toxicity data for risk assessment (benzyl alcohol), or availability of prior MCCEM exposure simulations

(NMP). Special considerations for relative potential acute health risks from use of these three alternatives are discussed in detail in Section 4.2.2 of this report.

Prior to running the alternative compounds exposure simulations, CTEH changed three MCCEM parameters to reflect the physical and stripping properties of each compound: chemical mass release rate, performance-equivalent mass applied and residence time. The values selected for amount applied and residence time for each MCCEM simulation were determined based information from the WM Barr (2015) alternatives performance assessment study. The chemical release rates were calculated based on previous calculations for MeCl2 and evaporation properties for each compound. The parameter value selection process is described in detail in Section 4.2.1.

CTEH also performed searches of the scientific literature to identify toxicology data to define single or short-term exposure PODs for each of the alternative compounds. Data that identified a threshold for acute toxicity from inhalation exposures of similar duration to the MCCEM simulations were preferred; however, professional judgement was used for selection of the most appropriate data set and POD. For each POD identified, a benchmark MOE was determined by CTEH as was done by USEPA (2014d) for MeCl2. Each benchmark POD captured the aggregate scientific uncertainty associated with extrapolating the POD to potential short-term human exposures. The identified PODs and benchmark MOEs are described in Section 4.2.2 of this report. Once PODs, benchmark MOEs, and MCCEM-simulated exposure estimates were determined for an alternative compound, a MOE was calculated as the POD divided by the MCCEM maximum average exposure estimate over a time period (hours) commensurate with the exposure duration associated with the POD data, as was done by USEPA (2014d). Using EPA's criteria for "unreasonable risk", a calculated MOE that was less than the compounds benchmark MOE was noted as potentially presenting an unreasonable risk for adverse effects. In this way, CTEH performed a parallel assessment of the relative acute risks of alternative compound uses that is directly comparable to EPA's MeCl2 assessment.

2.0 Comment #1: EPA employed a Multi-Chamber Concentration and Exposure Model (MCCEM) that overstates consumer exposures during residential use scenarios.

The Agency's risk assessment for MeCl2 and NMP consumer use paint stripper products "*…relied on a model-based consumer exposure assessment in the absence of sufficient measured data for consumer exposures to DCM-based paint strippers*" (USEPA, 2014d). The exposure scenarios modeled by EPA drew from published reports on home size and ventilation characteristics, as well as paint stripper uses and some reports of certain user behaviors while using strippers. However, EPA did not include in its exposure modeling and subsequent risk calculations other user behaviors reported in the same literature that represent a significant portion of consumer paint stripper users, and EPA did not take into account product efficacy considerations that will significantly affect user behavior and exposure scenarios.

The Agency's consumer exposure model exaggerates the likely exposures that consumers who use paint strippers in residential use setting actually experience. The exposure estimates do not reflect a reasonable maximum exposure representative of such uses, do not encompass likely exposure scenarios, and are inconsistent with the best scientific methods and EPA's scientific standards. These deficiencies were

further exaggerated in the Agency's 2016 supplemental exposure analyses, which involved use scenarios selected to produce higher consumer exposure and risk estimates. (The resulting supplemental analyses of consumer exposures for MeCl2 and NMP paint strippers have not been subjected to peer review.) The 2016 updates were deliberately designed to reflect additional use scenarios "with extended application times, increased product use and altered user behaviors." Following are some examples of the numerous ways in which the original consumer exposure scenarios EPA modeled for MeCl2 and NMP, and the expanded scenarios modeled in 2016, do not reflect reasonable assumptions about consumer use:

- The predominant location in which paint stripping work is typically undertaken by residential users is outdoors (CPSC 1992 Survey; Reilly 2001). None of the scenarios EPA modeled assumed outdoor uses. The inclusion of outdoor stripping scenarios would have given a more complete representation of actual user behaviors with paint strippers than did the limited number of scenarios used in the MeCl2 and NMP risk assessments and supplemental exposure assessments. The range of resulting MOEs, both from indoor and outdoor uses, would allow for a more informed consideration of the propensity for users to engage in uses in which unacceptable risks may occur.
- Several scenarios modeled do not reflect the product user leaving the work area during wait times (time between application and scraping for stripper to react with coating). The MeCl2 main risk assessment (Scenario #2 and #5), MeCl2 supplemental exposure assessment (Case ID #C4 and #F4), NMP risk assessment (Case ID #2, #6A, and #6b), and NMP supplemental exposure assessment (Case ID # C3 and #F4) simulate user exposures in which the user remains in the same room as the paint stripper-treated project during multiple 15-minute waiting periods ranging cumulatively from 30-60 minutes. Yet survey data EPA references show that the majority (60% according the survey of Riley et al. (2001) and greater than 50% according to CPSC (1992) of consumers rapidly leave the location where a paint stripper is used both during breaks and immediately after the work is completed (EPA 2014 MeCl2 Risk Assessment Appendix H). The EPA-simulated maximum continuous user time spent working with paint stripper on the dining room table/chairs scenario, without leaving the work area, was 154-198 minutes for MeCl2 and 196-225 minutes for NMP. This is in stark disagreement with the CPSC (1992) survey in which the majority of respondents worked for no more than 21 minutes on a project before taking, on average, a 10-minute break. The dramatic difference between these simulated and reported continuous work times casts doubt on the validity of the table/chairs scenario in which users did not leave the work area until a complete application and removal of stripper was finished for the table and eight chairs. Further, the effect of 10-minute breaks in between 21 minute work periods, as reported by CPSC, would significantly limit the peak user blood levels of MeCl2 or NMP, for these scenarios and likewise limit the time-average inhalation exposure values used in the EPA risk calculations. The likelihood of user taking breaks without washing from their hands of a coating of NMP is also low. The actual use of more frequent breaks in project task segments would obviate the continuous NMP dermal exposure modeled by EPA, and would have significantly reduced PBPK-modeled NMP blood levels as well as the associated risk calculations.
- The amount of product estimated to be used in the modeled scenarios was calculated to reflect median (50% of reported values) and an upper range (80%) value; no scenarios were run using

low-end use scenarios, as would be likely to be experienced when a consumer is using one of the more effective paint stripper product. In fact, no consideration for the relative efficacy of MeCl2, NMP, or other paint stripper ingredients was considered when determining the various task-specific time segments fir EPA MCCEM modeling. As shown below in the CTEH assessment of alternative compounds, the impact of stripper efficacy on exposure estimates is quite significant, and must be considered to properly use data to estimate exposures for specific stripper mixtures.

- EPA's 2016 Supplemental Assessment added a scenario for consumer stripping of an entire floor in 240 square foot room, yet EPA's consumer model assumed the space in which the floor would be stripped was within the much smaller work room itself, thereby over stating the likely airborne concentrations of paint stripper in the room and understating the air exchange rate. The EPA-defined work shop (Appendix H of the 2014 MeCl2 Risk Assessment (USEPA, 2014b)) area has a floor space of 120 square feet, or half of the stripped floor. A work shop with a floor space of 240 square feet would have a larger volume and would necessarily require a larger air exchange rate with the outside of the house in order to achieve the total house air exchange rates used in the EPA simulations.
- EPA has modeled consumer exposures during bathtub stripping, even though EPA's risk assessment specifically states that EPA assumes professionals perform bathtub stripping. (EPA 2014 MeCl2 Risk Assessment Appendix H.)
- EPA's model assumes all consumers will repeatedly reapply the paint stripper product. This effectively increases predicted exposures to consumers by as much as 2-fold. This assumption stands in contrast to testing information provided by Barr (2017) that reflects MeCl2 containing strippers, and certain NMP containing strippers will remove multiple layers of dried coatings with a single application.
- The model assumes residential users and bystanders will remain indoors for a 24-hour period including and following product use. However, consistent with the CPSC survey, consumers report they both scrape and dispose of the removed coating soon after the stripping step has been completed. EPA should have incorporated into its model the clean-up step occurring quickly following product application and stripping (especially for modeling consumer exposures to MeCl2 paint strippers which the Barr Report from 2015 show to perform more effectively, and more rapidly than other strippers). Clean-up activities result in increased ventilation in the work area and more frequent air exchanges throughout the residence, which will considerably reduce residual exposures to the paint stripper user, and bystanders.

Simulations of exposure scenarios in which the factors were better represented would have resulted in considerably lower (2-fold or more) estimates of consumer exposures and affected the agency's overall assessment of risk. A robust analysis of potential risks to a population of consumers using the best scientific methods would include the full range of potential exposures informed by all reasonably anticipated use behaviors, not simply worst case scenarios. This information would be helpful in determining if potential exposures resulting in unreasonable risk will occur, and if so, with what frequency. Such estimates also help inform risk mitigation decisions, such as issuing warnings and labeling products to advise against indoor uses, or use in certain low ventilation higher risk environments.

3.0 Comment #2: EPA relied upon candidate PODs from previously developed toxicity assessments that did not use best practices and methodologies available at the current time of the MeCl2 assessment in 2014.

As acute exposure to any chemical increases, a sequela of adverse effects will occur in a population, increasing in severity as the exposure level increases. This relationship of exposure to effect, known as the dose-response relationship, is a central tenet in toxicology. Within the dose-response continuum, the critical effect is defined in toxicology as the effect for a given duration and route of exposure that is most likely to occur first in a population as the exposure concentration increases. Consequently, the critical dose is the exposure level at which the critical effect is observed to occur. For acute, non-cancer toxicological effects, the critical dose represents the threshold demarking toxicity from non-toxicity. In chemical risk assessment, this is the chemical's POD for a given exposure duration.

The dose response of MeCl2 after inhalation in humans has been described in the scientific literature. Central nervous system (CNS) effects are the primary adverse health impacts from single or short-term MeCl2 exposures (ATSDR, 2000; NAS, 2009; USEPA, 2011). Effects from repeated, daily exposures are different from single or short-term exposures and result from repeated contact of MeCl2 with specific organ systems over long periods of time (USEPA, 2011). These exposure conditions and chronic effects are not relevant to potential acute exposures from consumer uses because the sporadic nature of consumer uses does not give rise to chronic, repeat-exposure effects. Indeed, EPA (2014d) evaluated acute, but not chronic, health risks to consumers, stating *"The focus on acute exposures was based on the assumption that DCM is not expected to significantly build up in the body between exposure events."*, and *"EPA/OPPT assumed that consumers would not generally strip paint on a regular basis in their residences allowing sufficient time between exposures to clear DCM and its metabolites from the body."* MeCl2 affects the CNS directly by suppressing neurological function, or indirectly by being metabolized in the body to carbon monoxide (CO), which reacts with hemoglobin in the blood to produce carboxyhemoglobin (COHb), which lowers the amount of oxygen transported by blood to other tissues, including the CNS. MeCl2 itself, rather than a metabolite of MeCl2, is the putative toxicant for acute CNS effects (Schlosser et al., 2015).

Fatalities during bathtub stripping by professional contractors have occurred at very high inhalation exposures estimated to be in excess of 300,000 mg/m^3 (CDC, 2012). However, several studies of experimental MeCl2 inhalation exposures in human volunteers (summarized by NAS (2009): DiVencenzo et al., 1972; Stewart et al., 1972; Winneke and Fodor, 1976; Fodor and Winneke, 1971; Winneke, 1974; Putz et al, 1979; Gamberale et al., 1975; and Kozena et al., 1990) indicated transient depression in reaction times on standardized neurological tests, subjective reports of light-headedness, which disappeared rapidly after cessation of exposure, and elevated COHb blood levels following 30-minute to 4-hour exposures of 677 to 3,474 mg/m^3. Thus, direct observation of volunteers in controlled exposure environments did not identify MeCl2 exposures less than 3,474 mg/m^3 that affect consciousness or the ability to egress to fresh air.

EPA considered four different PODs taken from three independently-performed assessments of potential acute human toxicity from MeCl2 inhalation (NAS, 2009; NRC, 1996; OEHHA, 1999). Unlike the NMP TSCA Work Plan Risk Assessment (USEPA, 2015b), EPA did not derive its own POD for MeCl2, but rather relied on off-the-shelf assessments. That is, EPA considered previously-published exposure limits for acute-duration (1 to 8 hour) exposures to use as PODs for MeCl2 risks from paint and coating stripping. Although this process is used in the regulatory world (e.g., EPA Reference Doses used for lifetime public health risk assessment; Agency for Toxic Substances and Disease Registry (ATSDR) Minimal Risk Levels used for hazardous waste workers risk assessment), it does not reflect the best available science because better methods were available to derive a POD that is more specific to the exposure scenario of interest, as was done by EPA for NMP. EPA did not offer a reason for selecting among pre-existing exposure limits for MeCl2 when better science and methodology (as discussed below in Section 2.1.3) were available at the time.

The first of the four candidate PODs was derived by the NRC (part of the NAS). The Spacecraft Maximum Allowable Concentration (SMAC) for MeCl2 of 350 mg/m^3 is a 1-hour average level intended to protect astronauts from developing more than 3% blood COHb levels (NRC, 1996). This SMAC value was based on a statistical extrapolation of volunteer exposure data for a 4-hour exposure to 700 mg/m^3 that resulted in blood COHb levels of 5% and impaired hand-eye coordination to a 1-hour exposure that would result in a blood COHb no higher than 3%.

California's Office of Environmental Health Hazard Assessment (OEHHA) derived the second candidate POD, an acute Reference Exposure Level (REL) POD of 840 mg/m^3 for a 1-hour average exposure "*at or below which no adverse health effects are anticipated in a human population, including sensitive subgroups, exposed on an intermittent basis*." (OEHHA, 1999). The REL was based on volunteer data in which changes in neurological tests (concentration and auditory vigilance), as well as blood COHb levels of 5.5%, were reported following a 1.5-hour exposure to 696 mg/m^3.

The National Academies of Science (NAS) established Acute Exposure Guideline Levels (AEGLs) for us in emergency incident planning for protection of public health. There are three levels of AEGL values for chemicals, each representing an exposure level that may result in increasingly severe health effects. AEGL-1 values are airborne concentrations of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic, non-sensory effects. AEGL-2 values are airborne concentrations above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects, or an impaired ability to escape. AEGL-3 values are airborne concentrations above which it is predicted that the general population, including susceptible individuals, could experience life-threatening health effects or death. Each level of AEGLs have associated time-dependent average concentrations for 10-minute, 30-minute, and 1, 4, and 8-hour exposure durations.

The third and fourth candidate PODs came from the NAS derivation of AEGL-1 and AEGL-2 values for MeCl2. NAS (2009) derived AEGL-1 values of 1,000 to 710 mg/m^3 averaged over exposure durations from 30 minutes to 1 hour. AEGL-2 values were also derived, ranging from 6,000 mg/m^3 down to 2,258 mg/m^3 for exposure durations averaged from 30 minutes to 8 hours. The AEGL-1 values for exposures of up to 1-

hour are based on human data for acute CNS depression effects, while the AEGL-2 values are based on production of blood COHb, an effect that is manifested after approximately an hour of MeCl2 inhalation exposure (NAS, 2009).

The SMAC, REL, and AEGL values were all considered by USEPA (2014d) as PODs for use in the MeCl2 risk assessment. EPA calculated MOEs for potential single consumer use of MeCl2 based on both AEGL values, the SMAC, and the REL, but the Agency preferred the SMAC-based POD and ultimately relied on this POD as the basis for its proposed determination that MeCl2 paint strippers present an unreasonable risk to users. CTEH critique of this process and of EPA's preference for the SMAC-based POD is discussed in this section.

3.1 The SMAC-based POD and associated benchmark MOE are not relevant to consumer uses of MeCl2 for paint and coating stripping

Evaluation of the critical effects identified in the SMAC, REL, and AEGL assessments and the associated benchmark PODs employed by EPA indicates that the SMAC is the least preferable of the three assessments for POD selection for the following reasons. The OEHHA REL is based on acute subtle CNS impairment effects (hand-eye coordination, peripheral light response time, and slowing of an auditory vigilance task), as reported following a 90-minute exposure of volunteers to 696 mg/m^3 MeCl2 (Putz et al., 1979). The AEGL-1 and AEGL-2 values are based on PBPK model computer simulations of brain levels of MeCl2 that would not likely cause CNS effects (AEGL-1 and AEGL-2) or result in additional blood COHb levels to over 4% of background (AEGL-2). The SMAC-based POD is an exposure limit "*designed for healthy individuals, and reversible effects might occur but they are not expected to impair the astronauts' judgment or interfere with proper responses to emergencies.*" EPA (2014c). The SMAC of 350 mg/m^3 was derived to protect healthy astronauts from incurring blood COHb levels higher than 5%. EPA stated that "*SMACs are designed for healthy individuals, and reversible effects might occur but they are not expected to impair the astronauts' judgment or interfere with proper responses to emergencies.*" Given those criteria, selection of a POD that is protective of the general public would be preferable to using the POD based on a SMAC value.

The EPA (2014d) preferred benchmark MOE of 10 associated with a SMAC-based POD was based on limiting blood COHb levels to protect sensitive subpopulations. Specifically, EPA cited a study reporting early onset angina in exercising coronary heart disease patients when their COHb levels exceed 2% over background (Allred et al., 1991, as cited in EPA, 2014). Exposure to MeCl2 during paint stripping activities, including the exposure scenarios used in the EPA risk assessment, would not be under conditions of strenuous physical exercise. No data were identified by CTEH to suggest that consumer paint stripping with MeCl2 is a physically strenuous activity. Indeed, the effectiveness of MeCl2 results in minimal physical exertion, compared to use of other compounds (WM Barr, 2017). Thus, EPA preferring a SMAC-based POD with the associated benchmark MOE of 10 for its potential acute consumer exposure risk assessment is not representative of adverse effects that may potentially occur during non-exertional use of MeCl2.

3.2 The approach used in deriving the AEGL-based PODs is more scientifically rigorous than, and preferable to, methods used to select the SMAC and REL-based PODs

Of the three previously developed hazard/dose-response assessments considered by EPA in the MeCl2 risk assessment (USEPA, 2014d), the technical approach used by NAS for deriving AEGL-1 and AEGL-2 values is scientifically preferable to the approaches used to derive the SMAC or REL values. Both the SMAC and REL PODs are based on observation from experimental exposures at a specific duration extrapolated to different time points. EPA stated that the typical total exposure duration for consumers using paint stripper is two hours (USEPA, 2014d). Appendix H of the EPA 2014 risk assessment shows MCCEM-simulated exposure profiles for consumer MeCl2 uses of approximately two to four hours. The SMAC value was based on observed effects from a 4-hour exposure that were extrapolated to estimate effects from a 1-hour exposure. Likewise, OEHHA (1999) derived the 1-hour REL from human data from 90-minute exposures. Further, the REL was based on an exposure that reportedly produced adverse effects, necessitating use of a higher benchmark MOE by EPA to account for uncertainty between the actual acute NOAEL and LOAEL of MeCl2. In both cases, the SMAC and REL values are derived for 1-hour exposures and applied by EPA to consumer exposure scenarios of two to four hours.

Use of a PBPK model to derive the AEGL values allows for estimation of average brain, blood, and air levels of MeCl2 at different exposure durations that are specific to potential exposures. This is one of the advantages of using well-developed PBPK models in human health risk assessment: estimates of chemical levels in blood and target tissues from observed exposures can be extrapolated to other exposure situations to determine the external air concentration necessary achieve the same internal dose and, presumably, the same adverse effect. EPA and NAS have previously recognized the use of PBPK models for this purpose (NAS, 2009; National Research Council, 2007; USEPA, 2014b); these types of models have been used by the Agency to derive chronic human toxicity values for multiple chemicals in the past, including MeCl2 (USEPA, 2011). EPA did not explain why its selection of the SMAC-based POD as preferable to the AEGL-based PODs, particularly with the use of better exposure extrapolation and POD determination capabilities afford by using a PBPK model.

It should be noted here that EPAs NMP TSCA Work Plan risk assessment (USEPA, 2017) utilized PBPK models for both lab animals and humans to (1) accurately simulate the dose-response behavior for NMP in pregnant rats, (2) extrapolate that toxicological relationship to humans, and (3) estimate a concordant human combination of dermal and inhalation exposure toxicity threshold in pregnant or childbearing-aged women. This same approach was used by EPA to extrapolate rodent toxicology study results to human chronic, lifetime exposure levels for MeCl2 that would produce an acceptable risk level. A similar approach could have been used for extrapolating experimental human study data to potential single or short-term exposure scenarios and identifying the associated PODs. However, EPA does not address or explain why it chose to use PBPK models for NMP but not for MeCl2.

3.3 EPA should have used the available and most advanced human PBPK model for use in deriving a single or short-term POD as it did for chronic exposures in the 2011 IRIS assessment.

As discussed in Section 3.2, use of a PBPK model for determining a MeCl2 POD would be preferable to the more generic and simplistic NOAEL/LOAEL approach used in determining the SMAC- or OEHHA REL-based PODs. Indeed, the PBPK model-derived NAS AEGL-1 and AEGL-2 values as POD surrogates are scientifically preferable. However, EPA failed to consider a human PBPK model for use POD derivation. In the IRIS assessment of MeCl2 (USEPA, 2011), EPA used a modified version of the David et al. (2006) PBPK model for MeCl2 to derive chronic cancer and non-cancer human toxicity values [Reitz et al. 1997 as cited by NAS (2009)]. This model is preferable to the model used in the NAS AEGL assessment for several reasons. First, it was calibrated based on human population and individual data on MeCl2 pharmacokinetics, accounting for both scientific uncertainty and experimental variability inherent in the individual model parameters. Thus, the model predictions of MeCl2 levels in the body following exposure were improved significantly over previous models (David et al., 2006; USEPA, 2011).

Second, the modified model used by USEPA (2011) for the 2011 IRIS assessment used probabilistic parameter distributions of model parameter values rather than deterministic point estimates. Briefly, a PBPK model defined by point estimate parameter values utilizes a single value for each model parameter. A given model simulation is based on a hypothetical individual having a single body weight, breathing rate, metabolic rate, etc. Such deterministic model predictions cannot convey the effect that variability within a population may have on the dose outcome. A probabilistic PBPK model, such as the modified David et al. (2006) model used by USEPA (2011) does not use single values for each model parameter, but instead uses statistical distributions of values to simulate a hypothetical population rather than an individual. Such distributions are a collection of values bounded by conditions such as a minimum, maximum, and average values. For example, a distribution for adult body weights may be bounded by a minimum of 45 kg, a maximum of 140 kg, and an average value of 80 kg. The PBPK model, then, is run repeatedly for many iterations (typically thousands) in which, for each iteration, a value is randomly selected from each of the parameter distributions and the simulation is run, thus defining a random individual described by that specific set of parameter values. The resulting prediction of MeCl2 internal or external doses is saved, and the next iteration is run. After many iterations, the collection of saved outcomes themselves define a distribution of internal and/or external doses for the entire hypothetical population. The characteristics of the dose based on the calculated distribution inform the effect on parameter variability, such as various body weights or metabolic capacities, on the predominant and fringe values of resulting doses. The use of this model, as was done for the 2011 IRIS assessment, would have resulted in more scientifically-robust PODs based on our best knowledge of the biology relevant to MeCl2 pharmacokinetics in the body and accounting for the effects of variability within a human population.

Third, the use of probabilistic parameter distributions for model parameters governing MeCl2 metabolism and COHb production allow for better resolution of internal dose metrics that may impact more sensitive subpopulations, such as people with coronary heart disease. The use of probabilistic parameter distributions for risk assessment in general has been endorsed by EPA (USEPA, 2014c). The EPA-modified

model of David et al. (2006) allows for the prediction for a range of blood MeCl2 and COHb levels for a population. This modeled population includes individuals with unusually high COHb production rates or low MeCl2 metabolism and clearance rates. These predictions allow for a better estimation of ranges (rather than single point) of external doses (e.g., inhaled MeCl2 concentrations) that may result in specific levels of blood MeCl2 or COHb. Use of a range of internal doses to define a POD such as COHb is preferable to relying on a single exposure level from a single study, as was done by EPA in applying the SMAC-based POD to exercising cardiac disease patients.

As stated in Section 1.2 of this report, EPA has committed to using the best scientific practices to underpin its regulatory decisions (USEPA, 2014a; USEPA, 2015a). Indeed, the use of the best-developed PBPK models for human health risk assessment aids significantly in addressing biological differences between species and physiological/biochemical variability with a species. Bois et al. (2010) indicated that the use of PBPK models, particularly population-level models like the one used in the EPA MeCl2 IRIS assessment, are well suited for understanding variability and reducing uncertainty in risk assessment. Breckenridge et al. (2016) also demonstrated the significant impact that PBPK modeling can have on reducing uncertainty in chemical risk assessment. Troutman et al. (2015) demonstrated how application of PBPK modeling methodology reduced uncertainty in a specific chemical risk assessment four-fold compared to default methods. EPA has stated that *"a PBPK or other TK [toxicokinetic] model provides the most biologically appropriate approach for evaluating intraspecies…extrapolation."* EPA (2014b). The use of a well-developed probabilistic PBPK model for human health risk assessments is a vital tool and represents the current and best methodology to reduce scientific uncertainty of deriving human toxicity values. These models can significantly reduce the chance of deriving under-protective or, conversely, over-conservative toxicity values that do not provide a sound basis for effective risk reduction measures.

4.0 Comment #2: EPA's POD, benchmark MOE, and exposure scenario selection introduced unnecessary scientific conservatism into its estimation of acute health risks.

For some well-studied chemicals, the critical effect and associated toxic threshold are well understood. For others, there may be uncertainty as to the identity of the critical effect or the threshold dose for that effect. All populations of living organisms have inherent variability with respect to the sensitivity of each individual to experiencing a chemical's critical effect (USEPA, 2014c). Some individuals will experience adverse effects at lower doses than less sensitive individuals. In chemical risk assessments, this uncertainty of a population-level threshold exposure and variability of individual sensitivity is dealt with by estimating a central estimate POD and applying UFs to account for uncertainty from extrapolating across species, routes of exposure, exposure durations, and variability within a human population. This results in an exposure level that is unlikely to cause the critical effect to any member of a population (NRC, 1983; USEPA, 1989).

4.1 EPA's Preferred SMAC-based POD brings an overly conservative benchmark MOE into the calculation of MOE risk estimates.

By not using the best available science and methodologies for POD determination, as discussed in Section 3.1 and 3.23 of this report, EPA has utilized benchmark MOEs that carry unnecessary uncertainty and conservatism through the rest of the quantitative risk assessment process. The benchmark MOE represents the level of variability and uncertainty in the POD's representativeness for a population. The magnitude of the benchmark MOE value determines the minimum fold-difference below the POD that an exposure value must be in order to be considered "reasonably safe" (Aylward et al., 2008; USEPA, 2013). For the four PODs considered by EPA in the MeCl2 risk assessment, there was an associated benchmark MOE that ostensibly accounted for the uncertainty in the underlying data and variability in population sensitivity for the respective POD's critical effect (Table 3-9 of USEPA (2014d)). The OEHHA 1-hour REL-based POD of 840 mg/m^3 has an associated benchmark MOE of 60, which includes UFs of 6 for using a LOAEL instead of a NOAEL, and 10 for variability of effect in sensitive humans. The SMAC-based POD of 360 mg/m^3 has an associated benchmark MOE of 10, which includes an UF of 10 for variability of effect in sensitive humans. The AEGL-1 PODs of 2,130 to 3,000 mg/m^3 for 30-minute to 2-hour exposures have an associated benchmark MOE of 3, which includes a UF of 3 for variability of target tissue effect in sensitive humans. Finally, the AEGL-2 PODs of 210 to 6,000 mg/m^3 for longer than 2-hour exposures have an associated benchmark MOE of 1, because the use of a PBPK model to extrapolate observed human effects data accounts for pharmacokinetic variability in humans, and because the data underlying the POD are from the most sensitive human population for COHb-mediated CNS effects (coronary disease patients). Thus, EPA's use of a benchmark MOE of 10 associated with the Agency's preferred SMAC-based POD would have been obviated if one of the PBPK model-based AEGL values had been utilized, and thus using this POD creates an unnecessary level of conservatism that cascades throughout the risk assessment.

4.2 Use of the most recent human PBPK model would allow for a lower benchmark MOE and less uncertainty in the risk assessment

The difference in UFs that defines the benchmark MOEs for the four different PODs considered by EPA is quite striking, considering that all four PODs are based on data for either acute CNS depression or CNS effects secondary to COHb production. The SMAC and REL-based PODs differed by about a factor of two (350 vs 840 mg/m^3) for application to 1-hour average exposure scenarios, with the SMAC representing the lower value. However, when the UFs were applied, the REL-based MOE was expanded by 6-fold over the MOE from the SMAC-based POD due to the fact that the underlying study design did not allow for identification of a NOAEL (the REL POD is based on the LOAEL of 840 mg/m^3). The resulting 1-hour exposure incurring low risk based on the REL would be 840 mg/m^3 ÷ 60 = 14 mg/m^3. Comparatively, the SMAC-based low risk exposure level is 350 mg/m^3 ÷ 10 = 35 mg/m^3. Comparing both of these low risk exposures with those of the AEGL-1 and AEGL-2-based PODs for 1-hour exposures (710 mg/m^3 and 667 mg/m^3, respectively), it becomes apparent that the POD selection/derivation itself can bring significant (order of magnitude) variation in low risk exposure levels. This illustrates the need to apply the best science to POD selection and address scientific uncertainty and population variability. Clearly, the use of a PBPK model to extrapolate an observed effect (COHb production and CNS depression) from a study to

various exposure durations in humans, and the inclusion of the biology of MeCl2 metabolism and elimination into the exposure calculation, results in a POD, its corresponding benchmark MOE, and low-risk exposure estimates that incorporate less uncertainty.

As described in Section 3.3, the application of the EPA-modified (USEPA, 2011) probabilistic PBPK model of David et al. (2006) for assessing potential acute consumer risks would utilize the best-developed methods for determining risk while reducing unnecessary uncertainty. As EPA said in 2006, "If the available human PBPK model is probabilistic in nature, accounting for the population distribution of parameters (biochemical, physiological, and physicochemical), the magnitude of the interindividual variability can be estimated." (USEPA and Office of Research and Development, 2006). The EPA-modified PBPK model for MeCl2 should be used by EPA to bring its risk assessment into line with its own technical recommendations.

4.3 Use of the best available PBPK model to derive a POD and associated benchmark MOE would result in lower consumer risk estimates than those proposed by EPA.

The use of best available science to define better benchmark MOEs would result in scenario-specific MOEs which may not indicate unreasonable risk for acute adverse effects to consumers using MeCl2 products. The most-advanced, vetted, and available PBPK model for MeCl2 should be used to simulate potential single or short-term inhalation exposures and derive a POD, taking into account (by virtue of the model's parameter distributions) variability in population physiology, metabolic capacity through the two prominent MeCl2 metabolism pathways (cyp2E1-mediated oxidation and GST-T1 conjugation), ethnic variations of these pathways, and variations in COHb production, and exposure duration-specific effects on internal doses of blood MeCl2 and COHb levels.

By utilizing a PBPK model that can provide population average and upper boundary levels of blood MeCl2 and COHb levels for a population, a POD value may be derived for a specific biological endpoint. For example, the model may provide an average airborne concentration of MeCl2 that results in a simulated COHb level for a sensitive subpopulation. A benchmark MOE of 1 would then be justified (as it was for the AEGL-2 POD reviewed by EPA), and would significantly lower the level of uncertainty in the resulting MOE risk estimates that would follow, compared to using the SMAC-based POD and its benchmark MOE of 10. The resulting PBPK-based MOEs may quantitatively indicate the lack of unreasonable risk for consumer use of MeCl2 for paint stripping purposes.

5.0 Comment #4 Use of alternative compounds instead of MeCl2 will not reduce acute health risks to consumer users based on analysis using EPA (2014d) methodology.

In the proposed rule, EPA stated that it "*has concluded that alternatives to methylene chloride…are available for nearly all paint removal uses.*" (EPA, 2017). In support of this decision, EPA developed a list of commercially-available paint strippers that did not contain MeCl2 (USEPA, 2016a). From that list, EPA a developed a list of 14 key compounds or compound classes (caustic hydroxides, dibasic esters) to

compare to MeCl2. The 14 ingredients/ingredient classes were briefly reviewed by EPA for toxicological effects, with NOAELs identified. Exposure potential relative to MeCl2 was characterized by briefly considering each compound's vapor pressure, molecular weight, and each ingredients' tendency to partition from water to non-polar solvents (an indication of dermal absorption potential).

CTEH compared the health risks of consumer uses of 8 of the 14 alternatives (used as single chemicals) as paint strippers with those of MeCl2 using the same methods employed by EPA for MeCl2. The three dibasic esters were simulated as a single compound. This included determining a toxicity POD and benchmark MOE for each alternative, performing MCCEM simulations of user and bystander exposure (for alternatives that may evaporate) as was done for MeCl2, calculating MOEs for each alternative, and comparing each MOE to its associated benchmark MOE. CTEH did not perform MCCEM simulations for benzyl alcohol (insufficient toxicity data available), caustic hydroxides (insignificant plausible inhalation exposures), methyl soyate (no toxicity data available), or NMP (exposure and risk assessment dealt with in Section 4.0).

5.1 Overview of EPA's assessment of alternatives (USEPA, 2016a)

USEPA (2016a) published an assessment of toxicity and exposure potentials for individual compounds selected as key ingredients for alternative products not containing MeCl2. This assessment was developed by Agency staff, but was never subjected to external scientific peer review. A list of 14 key ingredients for alternative products was presented and follows.

- Benzyl alcohol
- Dimethyl succinate
- Dimethyl glutarate
- Dimethyl adipate
- Methyl isobutyl ketone (MIBK)
- Methyl ethyl ketone (MEK)
- Toluene
- Acetone
- Methanol
- Calcium hydroxide
- Magnesium hydroxide
- N-methylpyrrolidone
- D-Limonene
- Methyl soyate

EPA treated dimethyl glutarate, succinate, and adipate as a combined class of dimethyl esters. EPA provided basic toxicity summary data for the compounds. This included NOAEL or LOAEL levels from published toxicity studies. The toxicity data presented for several compounds were from oral exposure or repeated dose toxicity studies utilizing exposure regimens not relevant to potential single or short-term consumer uses of these compounds for paint stripping. Thus, a robust comparison of relative toxicity of MeCl2 and the alternative compounds for potential short-term acute exposures could not be made except for the compounds for which data were summarized for rodent developmental effects from inhalation exposures over the relatively brief (days) period of rodent gestation.

In the alternatives assessment (USEPA, 2016a), the Agency grouped the 14 key compounds into six hypothetical product mixtures stated by the Agency to be representative of market products, based on a web-based search of paint strippers and their associated safety data sheets. Three of the hypothetical products were termed "baseline" NMP or MeCl2 products: two of these mixtures contained NMP; one contained MeCl2. The other three hypothetical alternatives contained various compositions of the non-NMP/MeCl2 alternatives. The Agency did not consider any specific effects of exposures to compound mixtures on consumer safety or toxicity. For each product mixture, the Agency presented a short synopsis of toxicity data from repeat exposures or long-term exposures that are not germane to consumer exposures. Further, the Agency did not provide an assessment of which, if any, of the mixture components would likely contribute the most to consumer health risks. No evaluation of ingredient additivity (multiple ingredients contributing to the same effect), antagonism (one ingredient countering the potential effect of another), or interdependence of effect was included. This gap limits the usefulness of the alternatives assessment to provide information on effects beyond those that may arise from exposure to single compounds only.

Several of the 14 key ingredients identified by EPA (2016a) are highly flammable materials, resulting in a physical health hazard that is not present for MeCl2. These ingredients include toluene, acetone, methanol, MEK, MIBK, and d-limonene (HSDB, 2017). Multiple paint stripper products have combinations of MeCl2 and other ingredients, including those that are flammable (Exponent, 2017; USEPA, 2016a; WM Barr, 2017). Although these MeCl2-containing products also contain flammable ingredients, the replacement of MeCl2 in such formulations with higher concentrations of the same or the addition of different flammable components will result in an alternative product with a higher risk to consumers of thermal burn and smoke inhalation injuries related to ignition of paint stripper. EPA has not reported any quantitative evaluation of the health or physical injury risk from combustion of flammable paint stripper products. However, section 4.2 of this report suggests that use of alternative products to obtain the same paint stripping performance as MeCl2 may involve at least twice the mass of very flammable compounds left on painted/coated surfaces indoors for many hours. Both of these factors introduce risks of compound ignition and fire/smoke that would not result from use of MeCl2.

EPA (2016a) did not consider the impact of consumer use behaviors on exposure potential for any of the hypothetical mixtures. The Agency determined alternative exposure potential to be similar, greater, or less than that of MeCl2 based on physical/chemical properties only. The user behavior characteristics discussed in Sections 1.1 and 2.0 of this report could influence relative exposure significantly. For example, the odor profile of one product may influence consumers to tend toward outdoors or well-ventilated indoor use more so than for another, less odorous mixture. Further, the type and amount of vapor inhibitors added to a product would dramatically influence exposure potential of components of a product. The Agency's lack of consideration of the effect of vapor inhibitors on component release to the air makes the entire discussion in EPA's assessment of relative exposure potential to be of questionable relevance to real-world scenarios.

EPA's (2016a) discussion of relative exposure potential was not quantitative and of very limited value for purposes of risk assessment. For each alternative compound, EPA briefly described the properties affecting dermal absorption or evaporation from a treated surface and stated whether these properties

would render the compound's exposure potential more, less, or equal (within a factor of 10) to that of MeCl2. In the proposed regulation preamble (USEPA, 2017), EPA stated that "*Overall, while the efficacies of the substitutes are comparable to the efficacy of methylene chloride, none of the substitute chemicals already available has the level of toxicity associated with methylene chloride.*" This statement was based on the EPA (2016a) alternatives assessment, which did not address comparative acute effects from single or short-term exposures. In fact, EPA has not presented any quantitative analysis of exposure estimates arising from single or short-term consumer use of the alternative compounds, except for NMP. This is a significant data gap for the position that potential acute consumer exposures to the alternatives are less risky with respect to development of adverse effects than are MeCl2 exposures.

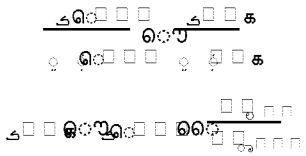
5.2 CTEH's performance-equivalence exposure estimation for alternative compounds

The purpose of the CTEH's MCCEM modeling of the alternative compound uses was to provide exposure estimates for MeCl2 performance-equivalent uses of the alternatives that are directly comparable to scenarios used by EPA to determine risks to consumers using MeCl2. CTEH used the MCCEM software to simulate user and bystander exposures using the same physical parameters (house volume, building ventilation rates, inter-zonal flow rates) used by USEPA (2014d) for an upper-end brush-on (EPA Scenario #6, described on page 250 of EPA [2014]) and spray-on (EPA Scenario #6, described on page 250 of EPA [2014]) product uses to strip a chest with a finished surface area of 25 square feet. CTEH selected EPA Scenarios #3 and #6 because they included parameters for the lowest house ventilation rate (0.18 air changes per hour) and the larger of the two pieces of furniture (clothing chest vs 10 square foot coffee table surface).

One of the key parameter values required for modeling the alternative compounds is the chemical release rate of the compound from the applied surface to the air. The MCCEM software uses a release rate constant in units of $hour^{-1}$, which is multiplied by the existing mass of compound to determine the rate released at a given time point. No data have been identified for the release rate of any of the alternative compounds. However, each compound has a known vapor pressure (at standard ambient temperature) that indicates the compound's tendency to change from the liquid to the gas phase and evaporate to the air. The higher the vapor pressure, the more likely and rapidly a compound will evaporate. Compounds in mixtures will be affected by the vapor pressures of other compounds within the mixture. For example, the addition of waxy vapor retardants to paint strippers or other solvent-based products during product formulation decrease the mass of compound that escapes to the air.

In the absence of chemical release data for the alternative compounds, CTEH calculated surrogate release rates based on each compound's vapor pressure and the relationship between MeCl2 vapor pressure and evaporation data developed by EPA. In order to use the MCCEM to estimate MeCl2 exposure to consumer users, USEPA (2014d) relied upon laboratory chamber data from MRI (1994) to statistically estimate a release rate. In the MRI (1994) study, a paint stripper-containing MeCl2 was applied to a surface in a controlled-environment exposure changer. An infrared detector instrument was used to measure MeCl2 concentrations in the air over time. EPA fit the chamber air concentration data to a statistical model to derive a release rate constant estimate of 10/hour, which was plugged into the MCCEM for MeCl2 use

scenarios. CTEH assumed that EPA's estimated MeCl2 release rate constant was a valid estimate and was directly proportional to the vapor pressure. Using this relationship, surrogate release rate constants for the alternatives were calculated using the following relationships,



where K_{Alt-} = Alternative compound release rate constant ($hour^{-1}$)

K_{MeCl} = EPA's fitted estimate for MeCl2 release rate constant ($hour^{-1}$)

VP_{MeCl} = MeCl2 vapor pressure of 435 mm Hg at 25 degrees Celsius

VP_{Alt} = Alternative compound vapor pressure at 25 degrees Celsius.

The resulting surrogate release rate constants are shown in Table 1.

Table 1: Alternative compound-specific parameters used for exposure estimation using the MCCEM

Alternative Compound	Vapor Pressure (mm Hg @ 25C)	Surrogate Release rate Constant (/hr)	Residence Time (minutes)	EPA (2014) exposure scenario	Total mass Used (grams)
benzyl alcohol	0.094	0.002	120	3	1,307
				6	1,952
dibasic esters	0.3	0.007	120	3	1,307
				6	1,952
acetone	231	5.310	120	3	1,307
				6	1,952
toluene	28.4	0.653	120	3	1,307
				6	1,952
methanol	127	2.920	120	3	1,307
				6	1,952
MEK	90.6	2.083	120	3	1,307
				6	1,952
MIBK	19.9	0.457	120	3	1,307
				6	1,952
d-limonene	2	0.046	120	3	1,307
				6	1,952
1,3-dioxalane	70	1.611	120	3	1,307
				6	1,952

In order to determine plausible performance-equivalent amounts applied and residence times for each alternative compound, CTEH used the WM Barr (2017) performance equivalence data for each of the

compounds. WM Barr (2017) reported on the ability of MeCl2 and a number of other single chemicals or paint stripper mixtures to allow for scrape removal of alkyd and epoxy paints or automotive paint finishes. (CTEH understands the 2015 and 2017 Barr Reports are included with Barr's comments.) For these tests, identical amounts of each compound were applied to surfaces coated with 5 layers of paint. Attempts were made to scrape off the paint after residence times of 15, 30, and 60 minutes. For alkyd paints, additional attempts were made to scrape off the paint after residence times of 4 and 24 hours. Removal performance at each time point was graded as one of four outcomes: (1) completed removal, (2) difficult removal with much effort, (3) slight paint softening without removal, and (4) no effect on paint. For epoxy paint, 1,3-dioxolane was the only one of the EPA-listed alternatives that successfully removed the paint, other than MeCl2. However, the reported residence times to sufficiently loosen epoxy paint were 15 minutes and 1 hour for MeCl2 and 1,3-dioxolane, respectively. These observations alone indicate that the alternative compounds suggested by EPA do not perform equivalently to MeCl2 and are not suitable replacements.

The study showed stripping performance on epoxy paints for up to 1 hour residence times. It is not known if the alternatives would be effective at longer residence times or after using higher amounts than MeCl2. CTEH conservatively assumed that the alternative compounds may effectively remove epoxy paint if amounts applied were twice that of MeCl2, and residence times were two hours. It is possible that even these use conditions would not result in effective paint removal. For the sake of comparative exposure modeling and risk comparisons, it is reasonable to believe that a consumer, upon not attaining satisfactory stripping of epoxy paint after one attempt, would apply twice the amount of an alternative (compared to MeCl2) and wait two hours to see if satisfactory results could be achieved. The total mass applied and residence times for each simulated exposure scenario are shown in Table 2.

For simulating exposure scenarios #3 (brush-on stripper applied to a clothing chest) and #6 (spray-on stripper applied to a clothing chest), EPA assumed that sections of the chest would be treated, 15 minutes residence time would be allowed for paint softening, and that the surface would be scraped in eight distinct phases, with half of the MeCl2 applied prior to a first scraping, and the process repeated with the remaining half of the total MeCl2 applied after the first scraping. CTEH followed this same convention, simulating half of the total amount applied to the entire chest over an 8-minute period (4 minutes for spray-on application), followed by a 2-hour residence time, then a 16-minute scraping and subsequent application of the second half of the total compound amount, a second 2-hour residence time for the second application, and a 16-minute final scraping thereafter. CTEH simulated the user exiting the workshop, but remaining in the house, during each residence time period. The MCCEM output for workshop concentration, user exposure, and bystander exposure levels for benzyl alcohol, toluene, acetone, methanol, dibasic esters, 1,3-dioxolane, MIK, MIBK, and d-limonene are provided in Appendix B. The sudden rise and fall of user exposure levels in the Appendix B figures are due to the user exiting and returning to the workshop area during wait time periods between scrapings. CTEH also assumed that the scrapings (including residual stripping compound that did not evaporate during the stripping procedure) were not removed from the workshop, but left for the remainder of the day. This is not an unreasonable assumption; however, removal of all remaining compound from the house after the second scraping (essentially eliminating the off-gassing source) would result in lower average exposures for compounds

with very low surrogate release rates (benzyl alcohol, dibasic esters, and d-limonene; see exposure profiles in Appendix B).

The MCCEM provided time-course air concentration estimates in 1-minute intervals. CTEH used these data for the user exposure to calculate running maximum averages for 1, 10, and 30 minutes and 1, 2, 4, 8, and 24 hours. The appropriate time-based average was paired with the PODs identified in Section 4.1.2 based on the exposure duration from which the POD was derived. For example, a POD from a study reporting a 4-hour exposure would be compared to the 4-hour average MCCEM exposure estimate; a POD from a study reporting a 6 or 7-hour exposure would be compared to the 8-hour average MCCEM exposure estimate.

5.3 POD and benchmark MOE selection for alternative chemicals

The scientific literature was searched and the identified acute inhalation exposure data were evaluated for the key alternative compounds identified by USEPA (2016a) to obtain relevant toxicity data and select appropriate PODs or lowest experimental doses associated with adverse effects in humans or animal models that fell within the range of exposures applicable to potential consumer exposures associated with single-project paint or coating removal at home. The study design, exposure route, duration, critical effect, and uncertainties associated with each POD are briefly described for each of the alternative compounds identified, as described below:

1,3-Dioxolane

A rat repeated-exposure inhalation study was conducted on groups of five male and five female Fischer 344 rats by exposing them to nominal concentrations of 1,3-dioxolane ranging from 0 ppm (control) to 5000 ppm. Animals were exposed for 6 hours per day, five days per week, for a total of nine exposures. Hematological effects were observed in rats exposed to 2000 ppm and 5000 ppm, but not in rats exposed to 500 ppm (ECHA, 2017; HSDB, 2017). Thus, the POD identified from this study was 500 ppm (1515 mg/m^3) as a NOAEL. To account for uncertainty resulting from interspecies differences, an Uncertainty Factor (UF) of 10 was applied. To account for intraindividual variability associated with human population heterogeneity (i.e. sex, age distribution, general health status, etc.), a UF of 10 was been applied. Cumulatively, the resulting benchmark MOE is 100.

Acetone

A human acute inhalation study conducted by Matsushita et al. (1969) on healthy male students evaluated the biological reactions associated with a 6 hour exposure (with 45 minutes' intermission in the middle) to various concentrations ranging from 0 ppm (control) to 1000 ppm acetone. Findings from this study showed that subjects exposed to 500 ppm or 1000 ppm acetone showed an increase in circulating eosinophil counts, as well as a reduction in phagocytic activity of circulating neutrophils. A slight decline in phagocytic activity was also observed in the 250 ppm group, but not in the 100 ppm group (Matsushita et al., 1969). Thus, the POD identified from this study was 100 ppm (237.5 mg/m^3) as a NOAEL. To account for uncertainty resulting from intraindividual variability a UF of 10 has been applied to the POD, resulting in a benchmark MOE of 10.

Dibasic Esters

A rat acute inhalation study conducted by Lee et al. (1992) evaluated the nasal irritation and lesions resulting from nose-only exposures to an aerosol/vapor mixture of dibasic esters at a concentration of 5900 mg/m^3 for 4 hours. Groups of rats exposed to this mixture were euthanized at various time points to evaluate recovery and regeneration of damaged olfactory and respiratory epithelium. The results from this study showed that at a concentration of 5900 mg/m^3, rats showed signs of damaged epithelium that regained normal appearance within 1-2 weeks; however, regions within the anterior nasal cavity resulted in more permanent damage that did not regain a normal structure after 6 weeks (Lee et al., 1992). Thus, the POD identified from this study was 5900 mg/m^3 as a Lowest Observable Adverse Effect Level (LOAEL). To account for uncertainty resulting from a LOAEL to a NOAEL, a UF of 10 was applied. To account for interspecies differences in a local, point-of-contact (nasal) effect, a UF of approximately 3.16 ($\sqrt{10}$) was applied, and to account for intraindividual variability, a UF of 3.16 ($\sqrt{10}$) was applied. Cumulatively, the resulting benchmark MOE is 100.

D-Limonene

A human acute inhalation study by Falk-Filipsson et al. (1993) evaluated pulmonary function, irritation, and symptoms related to central nervous system effects in healthy individuals during a 2-hour inhalation exposure to d-limonene at concentrations of 10, 225, and 450 mg/m^3. No reports of discomfort, irritation or central nervous system effects were observed for any of the test individuals up to the highest exposure concentration; however, a decline in vital capacity was observed following exposure to 450 mg/m^3 (Falk-Filipsson et al., 1993). Thus, the POD identified from this study was 225 mg/m^3 as a NOAEL. To account for uncertainty associated with intraindividual variability, a UF of 10 was applied to the POD for a resulting benchmark MOE of 10.

Methanol

A mouse repeated-exposure inhalation study conducted by Rogers et al. (1993) evaluated the developmental toxicity in pregnant female mice exposed to methanol concentrations ranging from 1000 to 15000 ppm for 7 hours per day between gestational days 6 and 15. Significant changes in number of cervical ribs were observed in the litter from mice exposed to 2000 ppm and higher methanol levels, in addition to increased incidences of cleft palates in litters of mice exposed to 5000 ppm and higher. No malformations on litters of mice exposed to 1000 ppm were reported (Rogers et al., 1993). Thus, the POD identified from this study was 1000 ppm (1310 mg/m^3) as a NOAEL. To account for uncertainty associated with interspecies differences, a UF of 10 was applied, and to account for intraindividual variability, a UF of 10 was applied. Cumulatively, the resulting benchmark MOE is 100.

Methyl Ethyl Ketone (MEK)

A mouse acute inhalation study conducted by Glowa and Dews (1987) evaluated the behavioral toxicity effects of MEK by measuring delays in scheduled controlled responses to a stimulus following 30 minute exposures to concentrations of MEK ranging from 885 to 29500 mg/m^3. No observable effects were reported when mice were exposed to 885 mg/m^3; however, at concentrations of 2950 mg/m^3, slight decreases in response rate were observed, with failure to respond observed at 16520 and 29500 mg/m^3 MEK. All response rates returned to baseline levels 30 minutes after exposure ended (Glowa and Dews, 1987). Thus, the POD identified from this study was 885 mg/m^3, as a NOAEL. To account for uncertainty

associated with interspecies variability, a UF of 10 was applied, and to account for intraindividual variability to a transient CNS effect, a UF of 3 was applied. Cumulatively, the resulting benchmark MOE is 30.

Methyl Isobutyl Ketone (MIBK)

A rodent sub-chronic inhalation study conducted by Tyl et al. (1987) evaluated the developmental toxicity in pregnant Fischer 344 rats and CD-1 mice exposed to methyl MIBK concentrations ranging from 0 (control) to 300 ppm during gestational days 6 through 15 (6 hours per day). Litters were evaluated for external, visceral and skeletal alterations, and clinical signs of toxicity such as decreased body weight and decreased food consumption were evaluated in dams. Reduced body weight and food consumption was observed in rats exposed to 3000 ppm. Similarly, reduced body weight in litters of rats exposed to 3000 ppm was observed. In mice, exposure to 3000 ppm resulted in increased maternal and fetal mortality as well as reduced fetal body weight per litter and reduced skeletal ossification. No observable adverse effects were reported in dams or litters of either species at concentrations of 1000 ppm or 300 ppm (Tyl et al., 1987). Thus, the POD identified from this study was 1000 ppm (4097 mg/m^3), as a NOAEL. To account for uncertainty associated with interspecies differences, a UF of 10 was applied, and to account of intraindividual variability, a UF of 10 was applied. Cumulatively, the resulting benchmark MOE is 100.

Toluene

A human acute inhalation study conducted by Anderson (1983) evaluated nasal and lung function, as well as irritation symptoms and neurological effects associated with exposures to toluene concentrations ranging from 0 ppm (control) to 100 ppm for 6 hours under controlled conditions. Irritation in the eyes and nose were reported by individuals exposed to 100 ppm, but no adverse effects were reported by individuals exposed to 10 or 40 ppm (Anderson, 1983). Thus, the POD identified from this study was 40 ppm (151 mg/m^3), as a NOAEL. To account for uncertainty associated with intraindividual variability in a point-of-contact irritation (eyes and nose) effect, a UF of 3 was applied, resulting in a benchmark MOE of 3.

Benzyl Alcohol and Methyl Soyate

No acute or repeated dose toxicity data are available for available for methyl soyate. The only inhalation toxicity data for benzyl alcohol is for lethality in laboratory rodents, which is not appropriate for calculating acceptable human risk level exposures. Thus, risk calculations for these two alternative compounds were not performed. MCCEM exposure estimates for benzyl alcohol were simulated for comparison with other alternative compounds, and are shown in Appendix B.

Caustics

Caustic alkalis, such as sodium hydroxide (i.e., lye or caustic soda), can be used as paint removers as they work to break down the chemical bonds of the paint through hydrolysis of the polymers resulting in a loosening of the paint from a surface. Caustic alkalis are typically non-volatile; sodium hydroxide has a reported vapor pressure of 1.82E-21 mm Hg at 25 degrees Celsius (NIOSH, 2016). Therefore, caustic alkalis were not evaluated using the EPA MCCEM model under the hypothetical in-home potential exposure scenarios developed by the EPA, and a direct comparison of risks associated with caustic alkalis to MeCl2 as a paint stripping agent was not conducted. It should be noted that the use of caustic alkalis as paint

strippers can cause dermal and ocular burns following brief contact (USEPA and Consumer Product Safety Commission, 1995).

Particulate Dust from Sanding

In addition to chemical-based paint strippers, non-chemical alternatives for removing paint and coatings from surfaces (i.e., wood, metal, and masonry) may include hand sanding and power sanding. EPA (2017) did not address sanding in the alternatives assessment (USEPA, 2016a), but did correctly note that inhalation of lead particles is possible if sanding is used to remove lead-based paint. Sanding does not typically utilize chemical paint strippers, thus the potential for consumer inhalation of chemical vapors is insignificant. Therefore, sanding was not evaluated using the EPA MCCEM model under the hypothetical in-home potential exposure scenarios developed by the EPA. Thus, a direct comparison of risks associated with sanding to using MeCl2 as a paint stripping agent was not conducted. Despite the lack of direct comparison to MeCl2, hand sanding and power sanding are not without the potential for risks to human health. While sanding is not associated with the production of chemical vapors, sanding of various surfaces has the potential to produce airborne particulate matter. Airborne dusts are of particular concern because they are associated with the potential to develop lung diseases based on the frequency, intensity, and duration of exposure (WHO, 1999). In addition to lung diseases, there is an increasing interest in other dust-related diseases such as cancer, asthma, allergic alveolitis, irritation, and non-respiratory illness that may occur at lower exposure levels (WHO, 1999).

Airborne particulate matter may consist of solid particles ranging from approximately 0.005 to 100 µm in aerodynamic diameter (Valavanidis et al., 2008). Particulate matter is generally classified according to its aerodynamic diameter, which is the primary determinant of evaluating the inhalation potential of an airborne particle (WHO, 2000). The type of sanding, the paint or coating being sanded, and the base surface from which the paint or coating is being sanded all add a level of variability with regard to the size, type, and chemical nature of the airborne particulate generated. For instance, the sanding of various materials including wood, metal, and masonry products can produce different airborne particulates. Wood dust or sawdust is a by-product of cutting, grinding, drilling, sanding or pulverizing wood or any other material with a saw or other tool; in other words, wood dust is defined as any wood particles arising from the processing or handling of woods (NIOSH, 2011). Wood dust exposure has been associated with a variety of adverse health effects. There are a large number of studies of humans with regard to wood dust, showing an association between dermatitis, allergic respiratory effects, mucosal and non-allergic respiratory effects, and cancer (NIOSH, 2016). The constituents of wood dust are variable based on the type of wood, working environment (e.g., tree cutting, saw mill, assembling, sanding), and other factors, and may include endotoxin, 1,3-β-D glucan, and solid and volatile components of wood (wood extracts) (ACGIH, 2016). Sanding of metals is usually a high energy process that can produce dust in a wide range of particle sizes. For example, sanding of steel alloys can contain metals such as beryllium, nickel, cobalt, chromium, lead, vanadium, and tungsten that can become airborne during sanding operations (WHO, 1999). Not only does the inhalation of airborne particulates have the potential to directly affect the respiratory system; but, the presence of particulate matter composed of metals such as lead, cadmium, beryllium, and manganese can be systemically absorbed into the blood stream and have been associated with effects on the blood, kidneys, and central nervous system. The severity of the potential for adverse

health effects is related to the type of heavy metal, its chemical form, and is time and dose-dependent (Gorguner and Akgun, 2010). Sanding of concrete or cement can generate airborne dust that can not only be inhaled but may directly affect the skin, causing various type of dermatitis (WHO, 1999). In addition, cement dust has been associated with lung function impairment, chronic obstructive lung disease, restrictive lung disease, and pneumoconiosis. In addition, exposure to cement dust is associated with irritation of the eyes, runny eyes and conjunctivitis, headache, and fatigue (Meo, 2004).

In addition to the base material from which the paint or coating is being sanded, the paint or coating itself can contain various constituents that can be present on airborne particulates following hand sanding or power sanding operations such as metals including lead, arsenic, cadmium, chromium, cobalt, copper, manganese, and nickel that can be associated with the potential for human health risks (Enander et al., 2002; Mielke et al., 2001).

5.4 Scientific uncertainties associated with acute toxicity or short-term exposure potential for alternative compounds preclude the ability to assess them as safe alternatives to MeCl2 for consumer use.

For each of the eight alternative compounds modeled using the MCCEM, CTEH divided the identified PODs by the exposure time-appropriate maximum average use concentration to arrive at a MOE. The resulting MOEs, along with benchmark MOEs for each POD, are shown in Table 2. All of the calculated MOEs are less than the associated benchmark MOEs. This table indicates that, using the same methodology used by USEPA (2014d), only one (dibasic esters) of the MOEs are greater than the associated benchmark MOE. Thus, the remainder of these compounds would be judged by EPA to result in an unreasonable risk of acute adverse effects on both users of these compounds and bystanders. Several alternative compounds (benzyl alcohol, methyl soyate, caustic hydroxides) and power sanding were not subjected to MCCEM simulation and/or MOE calculation because of data limitations discussed previously. However, Section 4.1.2 shows that the scientific uncertainties associated with the inherent toxicity and exposure potential for these compounds precludes the ability to assess them as being safe alternatives to MeCl2 for consumer use.

There are other consumer use scenarios for these alternative compounds that can be comparatively evaluated for health risk. The purpose of the present CTEH exercise was to leverage currently available performance and efficacy data (WM Barr, 2017) and standard EPA risk assessment methods (USEPA, 2014d) to determine whether these alternative compounds may provide a safer and equally effective substitute for MeCl2. Neither the alternatives assessment published by EPA, nor the analysis presented here, indicate that to be the case.

Table 2: Margins of Exposure for 8 Chemical Alternatives to MeCl2 Using MCCEM Exposure Estimates from Brush-on and Spray-on Paint Stripper Applications

Chemical Alternative	*Benchmark MOE*	*Comparable MCCEM Exposure Period a*	*EPA (2014) Scenario #3: Brush application in workshop, upper-end estimates*		*EPA (2014) Scenario #6: Spray application in workshop, upper-end estimates*	
			User	*Bystander*	*User*	*Bystander*
1,3-Dioxolane	100	4-hr max avg	2.6	2.7	1.8	1.8
Acetone	10	8-hr max avg	1.0	1.1	0.3	0.3
Dibasic Esters	100	4-hr max avg	164	164	110	110
d-Limonene	10	4-hr max avg	1.3	1.3	0.9	0.9
Methanol	100	8-hr max avg	2.3	2.3	1.6	1.6
Methyl ethyl ketone	30	8-hr max avg	2.7	2.7	1.8	1.8
Methyl isobutyl ketone	100	8-hr max avg	1.8	2.1	1.2	1.4
Toluene	3	8-hr max avg	0.3	0.3	0.2	0.2

[a] *Exposure estimates for MOE calculations were derived from maximum running concentration averages taken from MCCEM output for durations similar to toxicity study data on which PODs were based.*

6.0 Comment #5: EPA's 2015 NMP Risk Assessment and 2016 Supplemental Exposure Assessment apply overly conservative uncertainty and unreasonably high exposure estimates into the respective risk calculations.

USEPA (2015b) developed a TSCA Workplan risk assessment for NMP that evaluated risks from potential single consumer and single or short and long-term occupational uses as a paint and graffiti remover. For single or short-term consumer uses, EPA's approach to estimating inhalation exposure and calculating MOEs was the same as done for MeCl2 (USEPA, 2014d). A significant difference between the MeCl2 and NMP risk assessments, however, is the methods used to select a POD. While USEPA (2014d) considered four candidate endpoints identified from three different hazard/dose-response assessments for the MeCl2 risk assessment, the NMP assessment utilized a PBPK model for NMP in rodents and pregnant/child-bearing aged women to simulate the critical developmental toxicity study and extrapolate internal NMP doses across species.

The NMP rat model was used, basically, to convert administered doses reported in the critical rat study to blood levels. Benchmark Dose (BMD) modeling (not to be confused by the benchmark MOE) was then performed to identify a threshold for developmental effects that is statistically robust. The human PBPK model was then used to determine what aggregate dermal and inhalation exposures would result in the equivalent blood levels believed to not impute risk for developmental effects in humans. This process represents the use of best science to determine risk (Bois et al., 2010; Breckenridge et al., 2016; National Research Council, 2007; USEPA, 2014c; USEPA and Office of Research and Development, 2006). Using these methods, EPA (2015b) calculated MOEs for potential consumer exposures that were at or above the

selected benchmark MOE of 30. Thus, EPA's own analysis did not indicate high risk of adverse effects to a sensitive human subpopulation (pregnant women and their unborn children) from NMP uses that would most likely occur from using WM Barr products that contain NMP.

In 2016, EPA developed a Supplemental Exposure Assessment for NMP, which included paint stripping projects that were designed to result in much higher estimated exposures than those examined in the main 2015 risk assessment. Not surprisingly, the supplemental exposure scenarios resulted in exposures and associated risk MOEs that were determined by EPA to present unreasonably high risk to consumers. Details of specific flaws with EPA's Supplemental Exposure Assessment use scenarios were provided previously in Section 2.0 of this report.

6.1 Appropriateness of EPA's acute consumer benchmark MOE

EPA (2015b) derived a benchmark MOE of 30 based on two UFs. First, a UF of 3 was included to account for uncertainties in target tissue (the developing fetus) NMP sensitivity differences between pregnant rats and pregnant women. Use of this UF is justified in the absence of data indicating that the human fetus has the same or less sensitivity to NMP developmental effects.

Second, EPA (2015b) used a UF of 10 to account for potential variability in humans to both the pharmacokinetics disposition of NMP in the pregnant woman and variability in sensitivity of some women and fetuses to NMP's effects once it reaches the target tissues. EPA justified the UF of 10 by saying that the PBPK model does not account for variability in either pharmacokinetics or target tissue sensitivity. The PBPK model for NMP utilizes a single parameter estimate for NMP metabolism via the cyp2E1 oxidation pathway. EPA stated that, because there are data showing wide variability from person to person in cyp2E1 capacity, a single metabolism parameter value does not adequately address human pharmacokinetic variability. Further, EPA said that the PBPK model did not account for lifestage differences in NMP metabolism.

The reasons EPA gave for utilizing a UF of 10 for human variability are not substantiated by principle or historic precedent. The UF for human variability represents uncertainties in a population about whether some individuals are more sensitive to the effect of a compound on the target tissue (pharmacodynamic variability) or if some individuals absorb and retain the putative toxicant (the parent NMP molecule, in this case) in the body (pharmacokinetic variability). These sources of population variability are each apportioned approximately a factor of 3 of the overall UF of 10. The application of a UF of 3 for pharmacodynamics variability (sensitivity of the target tissues in some individuals) is warranted in the absence of data showing a lack of such variability in humans.

The application of a UF of 3 for pharmacokinetic variability is not justified. EPA concluded that a PBPK model utilizing a single point estimate for metabolic rate of NMP would not adequately account for population variability in metabolism. However, EPA failed to address how the resulting risk values might change if different (higher and lower) metabolic parameter values were used in the modeling. Use of the PBPK model, whose parameters are average values based on data from a range of values, does incorporate a level of human variability into the model output. The model also reflects dynamic biological

processes involved with the changing body of the pregnant women and fetus, resulting in an internal NMP dose likely to occur in pregnant women in general, a potentially sensitive subpopulation. Thus, use of the NMP PBPK model allows for reduction of the UF for interindividual pharmacokinetic variability to a value lower than 3.

Use of the PBPK model for assessing potential acute consumer risks from NMP use represents use of best available science and methodology, and serves to reduce scientific uncertainties incurred by extrapolation of a NOAEL across species and the application of arbitrary UFs. In the case of NMP, the cumulative UF, and benchmark MOE, should be 10 instead of 30. Together with more appropriate exposure factors that were discussed in Section 2.0 of this report, the corrected benchmark MOE of 10 would result in no unreasonable risk of acute adverse effects in consumers, and would also change EPA's determination of "high risk" involved with some occupational uses as well.

7.0 Summary and Conclusions

EPA intends to prohibit the manufacture, processing, and distribution in commerce of MeCl2 for all retail consumer use paint strippers and for most types of commercial paint and coating removal uses. Likewise, for NMP, EPA intends to either prohibit the manufacture, processing, and distribution in commerce of NMP for all consumer and commercial paint and coating removal products, or propose a reformulation, PPE, and labeling change approach to user risk reduction. These actions are based on EPA's TSCA Work Plan risk assessments for each chemical that identify unreasonable health risk to some or all users of paint stripping products containing MeCl2 or NMP.

CTEH evaluated EPA's technical approach and health-based bases for these decisions, and found significant shortfalls in the Agency's exposure and hazard assessment methods which affected its overall risk evaluation for potential risks to consumers who may use paint stripper products such as those made and sold by WM Barr.

7.1 CTEH conclusions regarding the 2014 MeCl2 consumer health risk assessment

CTEH found that EPA relied upon candidate PODs from previously developed toxicity assessments for MeCl2 that do not use best practices and methodologies available at the time of EPA's assessment. Of the three off-the-shelf hazard/dose-response assessments considered by EPA in the MeCl2 risk assessment (USEPA, 2014d), the technical approach used by NAS deriving AEGL-1 and AEGL-2 values that utilized a PBPK model is scientifically preferable to using SMAC or REL-based PODs and benchmark MOEs. In the IRIS assessment of MeCl2 (USEPA, 2011), EPA utilized a modified version of the David et al. (2006) PBPK model for MeCl2 to derived chronic cancer and non-cancer human toxicity values. The use of this model, as was done for the 2011 IRIS assessment, would have resulted in more scientifically-robust PODs based on our best knowledge of the biology relevant to MeCl2 pharmacokinetics in the body and accounting for the effects of variability within a human population.

Instead of using the best available science and methodologies for POD determination, EPA has instead selected benchmark MOEs that carry unnecessary uncertainty and conservatism through the rest of the quantitative risk assessment process. The use of a PBPK model to extrapolate an observed effect (COHb

production and CNS depression) from a study to various exposure durations in humans, and the inclusion of the biology of MeCl2 metabolism and elimination into the exposure calculation, results in a preferable benchmark MOE and low-risk exposure estimates that are backed by less uncertainty.

The use of best available science to define better benchmark MOEs would result in scenario-specific MOEs that would demonstrate that consumer use of MeCl2-containing products does not result in an unreasonable risk for acute adverse effects to consumers. A proposed benchmark MOE of 1 is justified. Associating this value with the AEGL-1-based POD would result in MOEs greater than the benchmark MOEs for all seven consumer exposure scenarios in the EPA risk assessment.

7.2 EPA used best available scientific methodology for the NMP risk assessment, but chose not to do so for the MeCl2 risk assessment

EPA's NMP TSCA Work Plan risk assessment (2015b) utilized PBPK models for both lab animals and humans, although the benchmark MOE selection was flawed. This same approach was used by EPA in the 2011 IRIS assessment for MeCl2 to extrapolate rodent toxicology study results to human chronic, lifetime exposures that would impart an acceptable risk level. A similar approach could have been used for extrapolating experimental human study data to single or short-term exposure scenarios and identifying the associated PODs. Although EPA stated that, "Our work will be guided by the best possible data and research and a commitment to transparency and accountability", the Agency's failure to use more advanced risk assessment technology that EPA used six years ago for the MeCl2 IRIS document casts doubt on the validity of this regulatory decision.

7.3 Quantitative risk assessment of alternative paint remover compounds does not show them to present comparatively more reasonable risks to consumers.

EPA's (2016a) discussion of relative exposure potential was not quantitative and of very limited value for purposes of risk assessment. EPA has not presented any quantitative analysis of exposure estimates arising from consumer use of the alternative compounds, except for NMP. This is a significant data gap; therefore EPA cannot support the position that consumer exposures to the alternatives present less risk for developing adverse effects than do MeCl2 exposures.

CTEH compared the health risks of consumer uses of 8 of the 14 alternative compounds or compound classes (used as single chemicals) as paint strippers with those of MeCl2 using the same methods employed by EPA for MeCl2. While other consumer use scenarios for these alternative compounds can be evaluated for health risk, CTEH used the same methods and exposure scenarios used by EPA to perform head-to-head comparisons of acute health risks to determine whether these alternative compounds may provide a substitute resulting in reasonable risk (as defined by EPA) to consumers. Neither the alternatives assessment published by EPA, nor the analysis presented here, indicate that to be the case. Based on the absence of any clear health risk advantage from the alternative paint stripper products, coupled with the list of flammability and combustion risks to consumer users of the alternatives as reported in the report prepared by Exponent (as included elsewhere in Barr's comments) it is difficult to find a reasonable and

defensible scientific basis to prohibit consumer uses of retail size MeCl2 and NMP-containing paint strippers.

To reflect the best scientific methods and the information reasonably available to the Agency, EPA must reevaluate its assessments of consumer risks to the retail size containers of MeCl2 and NMP paint stripper products. First, EPA must revise its modeling to more realistically and reasonably reflects consumer use scenarios -- including adaptations to reflect the predominant use of paint strippers occur outdoors and do not require repeat applications. Second, EPA must employ the latest probabilistic PBPK model for predicting hazards of MeCl2 exposures, using the methods the Agency used for its 2011 IRIS assessment of MeCl2 and the techniques it used for its NMP assessment. Finally, EPA must chose uncertainty factors (UFs) that are appropriate to the use of PBPK modeling, rather than the UFs EPA selected for MeCl2 and for NMP that are not necessary and reflect overly conservative values and overstate consumer risks.

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APPENDIX A: Curriculum Vitae of Michael H. Lumpkin, Ph.D., DABT

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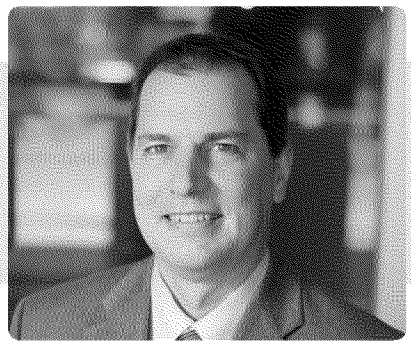

EXPERIENCE

Dr. Michael Lumpkin is a board-certified toxicologist with more than 14 years of experience in dose reconstruction, chemical dose response assessment, physiologically-based pharmacokinetic (PBPK) modeling, chemical emergency response, product stewardship and safety assessments, and litigation support. Michael has developed, critiqued and applied PBPK models for volatile organic compounds (VOCs), metals, pesticides and bioterrorism agents for USEPA, CDC and DOD, for use in regulatory standard support and emergency planning. He has developed a framework and performed safety assessments of dietary supplement products. He has coauthored numerous peer-reviewed hazard assessments for USEPA and the Agency for Toxic Substances and Disease Registry (ATSDR). He has provided critical reviews and analyses of toxicology data for numerous compounds, including formaldehyde, PAHs, inhaled dust and perchlorates. Michael has provided analyses of human and animal study data in support of new drugs and medical device applications, and has designed and performed occupational exposure reconstructions for VOCs and diisocyanates using laboratory simulations. He has developed novel occupational exposure limits for pharmaceutical and industrial chemicals. Michael has served on ad hoc federal grant review committees and as a peer reviewer for toxicology journals, and has lectured in graduate courses and emergency responder seminars for inhalation toxicology and risk assessment. He is a member of the Society of Toxicology and is certified as a Diplomate of the American Board of Toxicology.

EDUCATION

Ph.D., Toxicology	2002	University of Georgia Athens, Georgia
B.S., Biochemistry	1994	University of Georgia Athens, Georgia

REGISTRATIONS & CERTIFICATIONS

Diplomate, American Board of Toxicology – 2008, 2013
40 Hour HAZWOPER
8 Hour HAZWOPER Supervisor
TWIC Clearance
Firefighter I (NFPA 1001), National Board on Fire Service Professional Qualifications (2001)

PROFESSIONAL AFFILIATIONS

Member, Society of Toxicology (2003-Present)
Councilor, Risk Assessment Specialty Section of the Society of Toxicology (2014-2016)
President, Southeastern Regional Chapter of the Society of Toxicology (2011-2013)

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EMPLOYMENT PRIOR TO JOINING CTEH®

2010 - 2014	Senior Toxicologist / ENVIRON International Corporation, Arlington VA
2004 - 2010	Senior Toxicologist / Syracuse Research Corporation (now SRC, Inc.), N. Syracuse, NY
2002 - 2004	Toxicologist / Clayton Group Services (now Bureau Veritas), Kennesaw, GA
1994 – 1998	Research Coordinator / TRS Labs, Inc., Athens, GA

EXPERIENCE

Incident Response

- Served as the lead toxicologist of a team responding to a major crude oil unit train derailment and fire including interaction with U.S. Coast Guard, Federal and State regulators, local fire and law enforcement leadership, and community members.
- Served as the lead toxicologist of a team responding to a gasoline pipeline release and a petroleum refinery tank failure, including interaction with client environmental health professionals and members of the unified command.
- Provided toxicological support for a multidisciplinary team addressing chemical vapor exposures to workers at the U.S. Department of Energy Hanford nuclear site.
- Conducted air monitoring for multiple HazMat train derailments, petroleum pipeline releases, and industrial and natural gas well fires across the U.S.
- Provided toxicological and public health information to residents following a crude oil pipeline spill into the Yellowstone River in Montana.
- Provided toxicological support to railroad HazMat managers and emergency room attending physicians following worker chemical exposures resulting from a train derailment.
- Responded to and reported on health risk outcomes from a small-scale industrial chemical spill at a major aircraft manufacturing facility. The final risk assessment and communication report successfully allayed concerns within the exposed workforce for future health risks.
- Assessed likely health impacts of perchlorate exposures following an accidental release into a municipal drinking water system.
- Advised dietary supplement client on technical responses to alleged injury outbreak, including management of technical meetings with FDA and CDC investigators, and technical briefing of U.S. congressional staff.

PBPK Modeling and Pharmacokinetic Analysis

- Developed a rodent and human model for inhalation of benzo[a]pyrene for use in regulatory dosimetry and risk assessment.
- Developed rodent models for drinking water disinfection byproducts during pregnancy, and halogenated hydrocarbons for use in human health risk assessment.
- Developed prototype inhalation models for bioterrorism agents (anthrax and tularemia) in nonhuman primates capable of predicting internal doses deposited in the lungs and distributed through the lymphatic and circulatory systems.
- Developed preliminary PBPK models for inhaled polycyclic aromatic hydrocarbons (PAHs).
- Reviewed, critiqued and applied PBPK models for a variety of organic compounds including:

- Applied PBPK and benchmark dose models for acrylonitrile, dichloromethane and 1,4-dioxane in support of cancer and non-cancer dose-response chapters of USEPA IRIS toxicological reviews.
- Provided review and critique of PBPK models in support of USEPA IRIS assessments (vinyl acetate, carbon tetrachloride, tert-amyl methyl ether and n-butanol) and ATSDR toxicological profiles(benzene, ethylene glycol, vinyl chloride, phenol, perchlorate and diazinon).
- Provided analysis and interpretation of human pharmacokinetic data, including implications for drug safety, to pharmaceutical and dietary supplement manufacturers.
- Provided critical review and comparison of human pharmacokinetic data sets for combination oral contraceptive products.

Chemical Product Stewardship

- Provided toxicological support to a polystyrene manufacturer's investigation of customer-reported occupational dermatitis incidents.
- Developed a novel occupational exposure limit for a reaction product formed during manufacture of flexiblemedical tubing.
- Developed novel occupational exposure limits for cancer chemotherapy and testosterone-based drug products.
- Developed a novel occupational exposure limit for a manufacturing by-product compound using "read across" methodology.
- Developed multiple OSHA/GHS-complaint Safety Data Sheets (SDSs) for petrochemical and food products.
- Performed California Proposition 65 Safe Harbor analyses for a variety of consumer products.

Chemical Toxicity Value Derivation

- Provided updated dose-response assessments to clients based on newly available data for metals, VOCs and aldehydes.
- Developed evidence-based assessment of perchlorate drinking water standard and research priorities, presenting finding to an EPA SAB, at national scientific meetings, and to a major trade association.
- Provided critical comments pertaining to interpretation of toxicokinetic, mode of action and dose-response data for carcinogen toxicity assessments such as formaldehyde and PAHs.
- Co-authored toxicokinetics and dose-response chapters, including dose-response modeling of toxicity data to derive reference doses and concentrations, and cancer potency factors, for numerous USEPA IRIS toxicological reviews and peer-reviewed provisional toxicity value documents for solvents, metals and aldehydes.
- Authored toxicity chapters and derived non-cancer minimal risk levels for numerous ATSDR toxicological profilesfor VOCs, metals and pesticides.

Dose Reconstruction

- Derived dose estimates of asbestos fiber exposure for a variety of industrial settings.
- Designed and managed a laboratory dose reconstruction assessment of inhalation and dermal exposure to diisocyanates and other VOCs under simulated occupational conditions.
- Analyzed air sampling and exposure factor data to determine allowable PCB exposures in public school students.

Pharmaceutical Product Development

- Provided interpretation of human pharmacokinetic data in support of an FDA new drug application.
- Authored safety assessments of surgical implant devices in support of FDA approval for conduct of human trials.

Dietary Supplement Safety Assessment

- Developed a framework for client to use in study design and data interpretation for developing product-specificsafety assessments.
- Performed safety assessments for multiple dietary supplement products
- Helped client interpret pharmacokinetic data and design human clinical trials for assessing safety of high-cocao flavanol content supplements.
- Designed and monitored preclinical rodent toxicity studies for dietary supplement product ingredients.

Litigation Support

- Provided expert deposition testimony for a cases involving occupational ammonia, chlorine gas, and methylene chloride gas exposures, ethanol/drug pharmacokinetics and risk-based environmental health litigation.
- Provided risk-based analysis, expert reports, and expert deposition testimony regarding health impacts related to offsite groundwater VOC migration from a municipal landfill and residential pesticide exposures.
- Contributed to health impact assessment and expert report development in cases involving public exposures to bisphenol A, coal ash wastes, contraceptive hormones, landfillodors, as well as occupational exposures to Naturally Occurring Radioactive Materials (NORM), asbestos, 1,3-butadiene, diisocyanates, mixed VOCs and World Trade Center dust.
- Analyzed the effectof pharmaceutical incipients on gastrointestinal absorption and cellular effectsof an osteoporosis medication.
- Developed exposure modeling methods to compare disease risks over time in students and workers exposed to PCBs from aging lighting systems in a major metropolitan public school system.
- Developed exposure scenarios and risk-based exposure limits for PCBs in air of public school buildings.
- Assisted with future projections of population-level blood PCB levels in based on NHANES data.

PUBLICATIONS

A. Peer Reviewed Publications

1. Campbell, J, Franzen A, Van Landingham C, Lumpkin M, Crowell S, Meredith C, Loccisano A, Gentry R, Clewell C. 2016. Predicting lung dosimetry of inhaled particleborne benzo[a]pyrene using physiologically based pharmacokinetic modeling. Inhal Toxicol.28: 520-535.

2. Rodricks J and Lumpkin M. 2013. DMAA as a Dietary Ingredient. JAMA Intern Med. 173:594-595.

3. Rodricks J, Lumpkin M, Schilling B. 2013. Pharmacokinetic data distinguish abusive versus dietary supplement uses of 1,3-dimethylamylamine. Ann Emerg Med. 61:718-719.

4. Faroon O, Roney N, Taylor J, Ashizawa A, Lumpkin MH, Plewak, DJ. 2008. Acrolein environmental levels and potential for human exposure. Toxicol Ind Health. 24:543-564.

5. Faroon O, Roney N, Taylor J, Ashizawa A, Lumpkin MH, Plewak, DJ. 2008. Acrolein health effects.Toxicol Ind Health. 24:447-490.

6. Fisher J, Lumpkin M, Boyd J, Mahle D, Bruckner J, El-Masri H. 2003. PBPK Modeling on the Metabolic Interactions of Carbon Tetrachloride and Tetrachloroethylene. Environ Toxicol Pharmacol 16:93-105.

7. Lumpkin MH, Bruckner JV, Campbell JL, Dallas CE, White CA, Fisher JW. 2003. Plasma Binding of Trichloroacetic Acid in Mice, Rats, and Humans under Cancer Bioassay and Environmental Conditions. Drug Metab Disp 31(10):1203-1207.

8. Yu KO, Naarayanan L, Mattie DR, Godfrey RJ, Todd PN, Sterner TR, Mahle DA, Lumpkin MH, Fisher JW. 2001. The Pharmacokinetic of Perchlorate and its Effect on the Hypothalamus/Pituitary-Thyroid Axis. Toxicol Appl Pharmacol 181(2):148-159.

TEXT BOOK CHAPTERS

1. Lumpkin, MH. 2015. Chapter 58: Chlorinated Hydrocarbons. In Hamilton and Hardy's Industrial Toxicology. Ed. Harbison, RD.

2. Lumpkin, MH. 2015. Chapter 59: Other Halogenated Hydrocarbons. In Hamilton and Hardy's Industrial Toxicology. Ed. Harbison, RD.

PRESENTATIONS

1. Lumpkin M. 2014. Inhalation Toxicology for First Responders. Training seminar provided to firefighters, national guardsmen, and emergency medical technicians at multiple fire departments across North Dakota.

2. Lumpkin M, Crowell S, Franzen A, Gentry R, Kaden D, Meredith C, Potts, R. 2014. Development of a PBPK Model for Inhaled Benzo[a]pyrene in Rats and Humans. The Toxicologist 138:1. Presented at the 53rd Meeting of the Society of Toxicology in Phoenix, AZ.

3. Schlosser P, Lumpkin M, Morris J. 2013. Extension of a Nasal Dosimetry Model for Acetaldehyde to Account for Vasodilation. The Toxicologist 132:1. Presented at the 52st Meeting of the Society of Toxicology in San Antonio, TX.

4. Lumpkin M, Gentry P, Greene T, Shipp A, Cirone T. 2012. Reassessment of the Critical Effect of Perchlorate Toxicity in the Human Thyroid to Inform on Drinking Water Regulations. The Toxicologist 126:1. Presented at the 51st Meeting of the Society of Toxicology in San Francisco, CA

5. Lumpkin, M. 2011. Dose Reconstruction Inside and Out. Presented at the Fall Meeting of the Georgia Local Section of the American Industrial Hygiene Association in Atlanta, GA.

6. Lumpkin M. 2009. Developing Mechanistic Models for Risk Assessment of Biothreat Agents. Presented at the EPA-CDC Workshop on State-of-the-Science of the Determination and Application of Dose-Response Relationships in Microbial Risk Assessment. Centers for Disease Control in Atlanta, GA.

7. Lumpkin M, Diamond G, Massulik S, Coleman P. 2009. PBBK/BD Model of Francisella Tularensis in Rhesus Monkeys. The Toxicologist 108:1. Presented at the 48th Meeting of the Society of Toxicology in Baltimore, MD.

8. Diamond G, Lumpkin M, Rhoades J, Massulik S, Coleman P. 2008. Modeling Inhaled Microbes in Primates to Inform Discussions on "Acceptable Risk." Presented at the 2008 Annual Meeting of the Society for Risk Analysis in Boston, MA.

9. Lumpkin M, McClure PR, Diamond, G, Schlosser P, Cooper, GS. 2008. Assessment of Dichloromethane PBTK Model Performance in the Rat. The Toxicologist 102:1. Presented at the 47th Meeting of the Society of Toxicology in San Diego, CA.

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11. Keys DA, Lumpkin MH, Bruckner JV, Fisher JW. 2005. Incorporation of Trichloroacetic Acid Plasma Binding in Human and Mouse in Trichloroethylene Risk Assessment. The Toxicologist 84:1-S. Presented at the 44th Meeting of the Society of Toxicology in New Orleans, LA.

12. Lumpkin MH, Runnion V, LeickfieldR, Paul S, Harbison R. 2005. Simulation and Assessment of Occupational Exposures to Isocyanates and VOCs During Application of a Urethane Product Suite Under Worst-Case Conditions. The Toxicologist 84:1-S. Presented at the 44th Meeting of the Society of Toxicology in New Orleans, LA.

13. Lumpkin MH, Dahlstrom DL. 2004. Mold by the Numbers: The Strengths and Weaknesses of the Scientific Literature to Provide Mycotoxin-related IAQ Risk Assessment. Presented May 10, 2004 at the American Industrial Hygiene Conference and Exposition in Atlanta, Georgia.

PEER REVIEWED REPORTS

1. Lumpkin M, Plewak D. 2009. Toxicological Profilefor 1,3-Butadiene (Update, Draftfor Peer Reviewer Comment). Prepared for the Agency for Toxic Substances and Disease Registry.

2. Bosch S, Lumpkin M, Plewak D. 2009. Toxicological Review of Tert Amyl Methyl Ether (TAME, CAS No. 994-05-8) in Support of Summary Information on the Integrated Risk Information System (IRIS). (Internal EPA review). Prepared for the IRIS Program, National Center for Environmental Assessment, U.S. EPA, Washington, DC.

3. Lumpkin M, Odin M. 2009. Draftprovisional toxicity values for 4,6-Dinitro-o-cresol (CASRN 534-52-1). Prepared for the Superfund TechnologySupport Center, National Center for Environmental Assessment, U.S. EPA, Cincinnati, OH.

4. Lumpkin M, Odin M. 2009. Draftprovisional toxicity values for methyl acetate (CASRN 72-20-9). Prepared for the Superfund Technology Support Center, National Center for Environmental Assessment, U.S. EPA, Cincinnati, OH.

5. Lumpkin M, Odin M. 2009. Draft provisional toxicity values for 2-methoxyethanol (CASRN 109-86-4) and 2-methoxyethanol acetate (CASRN 110-49-6 and 32718-56-2). Prepared for the Superfund Technology Support Center, National Center for Environmental Assessment, U.S. EPA, Cincinnati, OH.

6. McClure P, Llados F, Osier M, Plewak D, Lumpkin M, Ellis B. 2008. Toxicological Review of Dichloromethane (Methylene Chloride) (CAS No. 79-09-2) in Support of Summary Information on the Integrated Risk Information System (IRIS). (Internal EPA review). Prepared for the IRIS Program, National Center for Environmental Assessment, U.S. EPA, Washington, DC.

7. Lumpkin M, Odin M, Carlson-Lynch H. 2008. Draftprovisional toxicity values for 2-methoxyethanol (CASRN 109-86-4). Prepared for the Superfund TechnologySupport Center, National Center for Environmental Assessment, U.S. EPA, Cincinnati, OH.

8. Lumpkin M, Odin M. 2008. Draftprovisional toxicity values for Diethylene Glycol Monoethyl Ether (CASRN 111-90-0). Prepared for the Superfund TechnologySupport Center, National Center for Environmental Assessment, U.S. EPA, Cincinnati, OH.

9. Lumpkin M, Chappell L, McClure P. 2007. Toxicological Profilefor Boron (Update, Draftfor Public Comment). Prepared for the Agency for Toxic Substances and Disease Registry.

10. Lumpkin M, Swarts S, Plewak D. 2007. Toxicological Profilefor Acrolein (Update, Final). Prepared for the Agency for Toxic Substances and Disease Registry.

11. Lumpkin M, Odin M, Carlson-Lynch H. 2007. Draftprovisional toxicity values for hydroquinone (CASRN 123-31-9). Prepared for the Superfund TechnologySupport Center, National Center for Environmental Assessment, U.S. EPA, Cincinnati, OH.

12. Lumpkin M, Odin M, Klotzbach J. 2007. Draftprovisional toxicity values for p-chloroaniline (CASRN 106-47-8). Prepared for the Superfund TechnologySupport Center, National Center for Environmental Assessment, U.S. EPA, Cincinnati, OH.

13. Stickney J, Llados F, Lumpkin M, Odin M. 2007. Toxicological review of 1,4-dioxane (CASRN 123-91-1) in Support of Summary Information on the Integrated Risk Information System (IRIS). Prepared for the IRIS Program, National Center for Environmental Assessment, U.S. EPA, Washington, DC.

14. Stickney J, Citra M, Lumpkin M. 2006. Toxicological profilefor vinyl chloride (Update, Final). Prepared for the Agency for Toxic Substances and Disease Registry.

15. Stickney J, Llados F, Lumpkin M, Odin M. 2006. Toxicological Review of 1,4-Dioxane (CAS No. 123-91-1) in Support of Summary Information on the Integrated Risk Information System (IRIS). Prepared for the IRIS Program, National Center for Environmental Assessment, U.S. EPA, Washington, DC.

16. McClure P, Llados F, Osier M, Plewak D, Lumpkin M, Ellis. 2006. Toxicological review of dichloromethane (methylene chloride) (CASRN 79-09-2) in Support of Summary Information on the Integrated Risk Information System (IRIS). Prepared for the IRIS Program, National Center for Environmental Assessment, U.S. EPA, Washington, DC.

17. Osier M Llados F Plewak D Lumpkin M Odin M (2006) Toxicological review of cerium (stable, CASRN 7440-45-1) and compounds in Support of Summary Information on the Integrated Risk Information System (IRIS). Prepared for the IRIS Program, National Center for Environmental Assessment, U.S. EPA, Washington, DC.

18. McDougal A, Wohlers D, Lumpkin M, McClure P. 2006. Toxicological Review of Mirex (CAS No. 2385-85-5) in Support of Summary Information on the Integrated Risk Information System (IRIS). Prepared for the IRIS Program, National Center for Environmental Assessment, U.S. EPA, Washington, DC.

19. Fransen M, Lumpkin M, Rhodes J, McClure P. 2006. Toxicological Review of Acrylonitrile (CAS No. 107-13-1) in Support of Summary Information on the Integrated Risk Information System (IRIS). (Internal EPA review). Prepared for the IRIS Program, National Center for Environmental Assessment, U.S. EPA, Washington, DC.

20. Llados F, Garber K, PaikoffS, Lumpkin M. 2006. Toxicological profilefor Phenol (Update, Final). Prepared for the Agency for Toxic Substances and Disease Registry.

21. Lumpkin M, Odin M. 2006. Draftprovisional toxicity values for bifenox (CASRN 42576-02-3). Prepared for the Superfund Technology Support Center, National Center for Environmental Assessment, U.S. EPA, Cincinnati, OH.

22. Wohlers D, Lumpkin M, Coley C, Hard C. 2006. Toxicological profilefor diazinon (Update, Final). Prepared for the Agency for Toxic Substances and Disease Registry.

23. Klotzbach J, Lumpkin M, Odin M. 2006. Draftprovisional toxicity values for bis(2-ethylhexyl)phthalate (CASRN 117-81-7). Prepared for the Superfund Technology Support Center, National Center for Environmental Assessment, U.S. EPA, Cincinnati, OH.

24. Lumpkin M, Odin M. 2006. Draftprovisional toxicity values for 1,1-dimethylhydrazine (CASRN 57-14-7). Prepared for the Superfund TechnologySupport Center, National Center for Environmental Assessment, U.S. EPA, Cincinnati, OH.

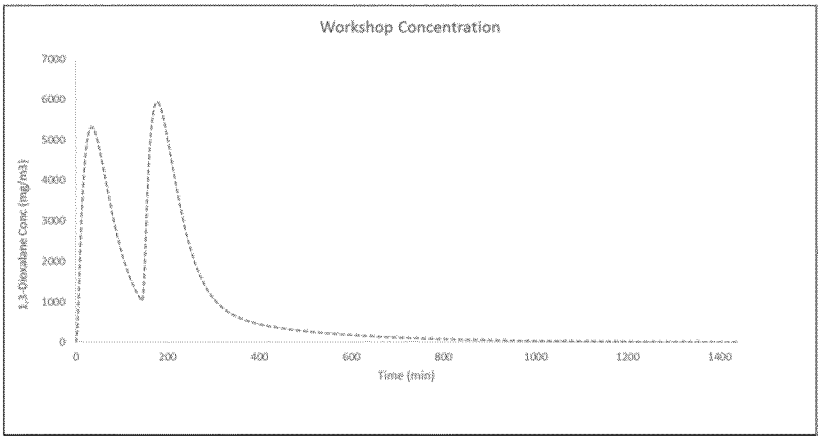
25. Lumpkin M, Ingerman L, Plewak J, Moilanen L, Beblo D, Walters J. 2005. Toxicological Profilefor Bromoform and Dibromochloromethane (Update, Final). Prepared for the Agency for Toxic Substances and Disease Registry.

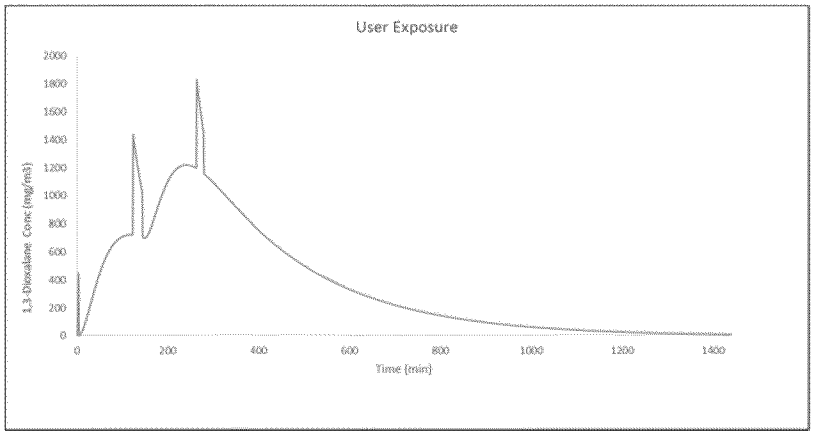
26. Bosch S, Citra M, Quinones-Rivera A, Lumpkin M, Rhoades J, Llados F. 2005. Toxicological Profilefor Alpha-, Beta-, Gamma-, and Delta-Hexachlorocyclohexane (Update, Final). Prepared for the Agency for Toxic Substances and Disease Registry.

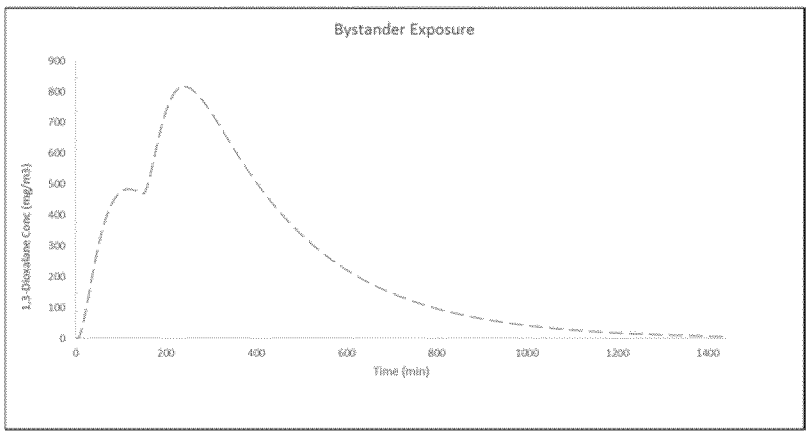
APPENDIX B: MCCEM Output for User and Bystander Exposures During Use of Alternative Compounds for Brush-on or Spray-on Paint Stripping Applications

1,3-Dioxolane

Scenario #3: Brush-on application

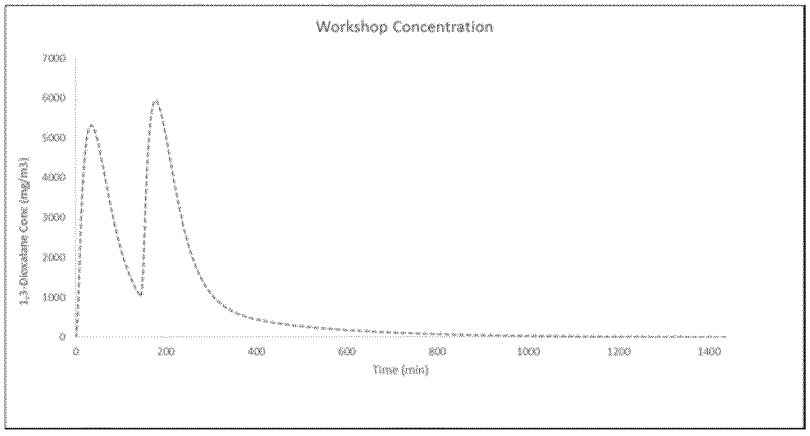


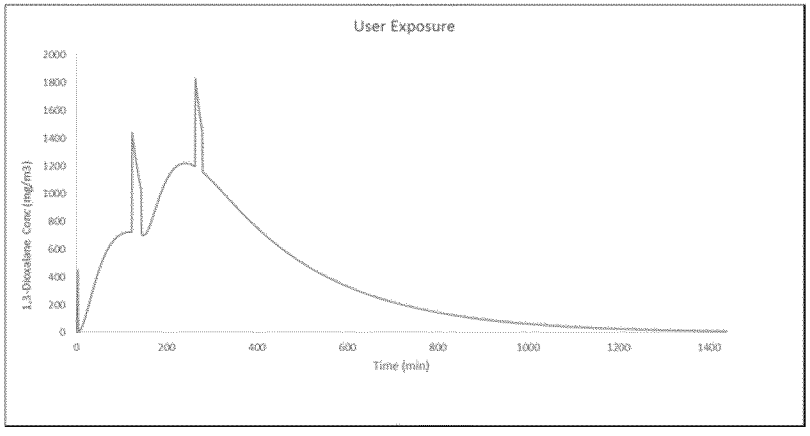


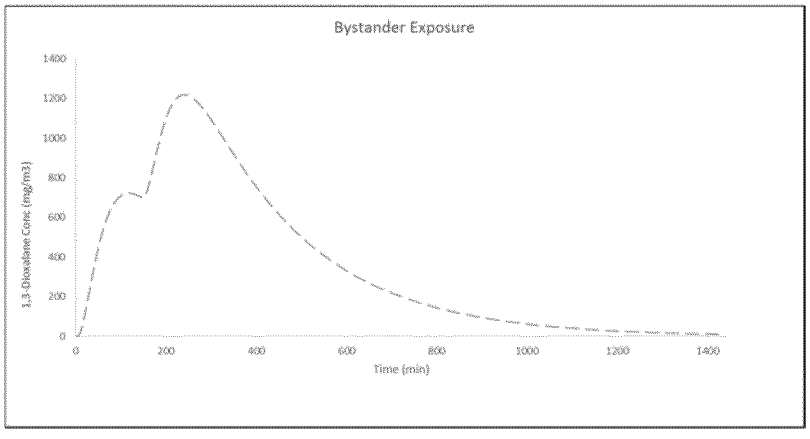


1,3-Dioxolane

Scenario #6: Spray-on application

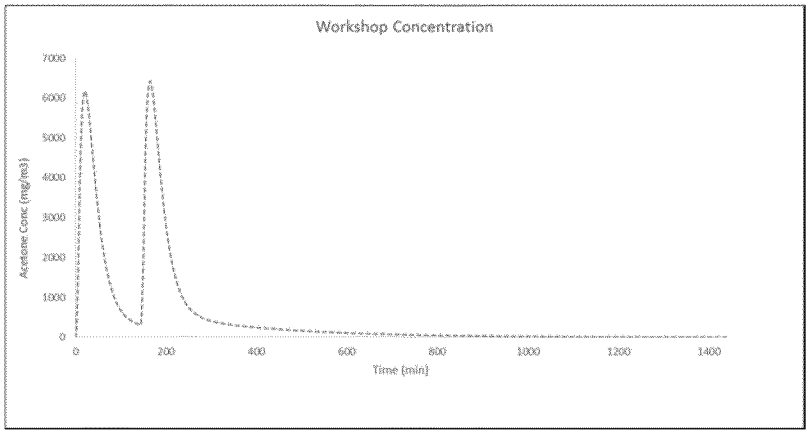


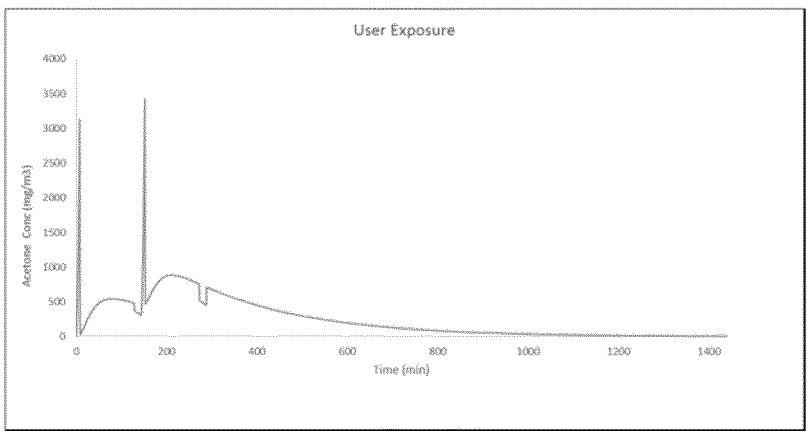


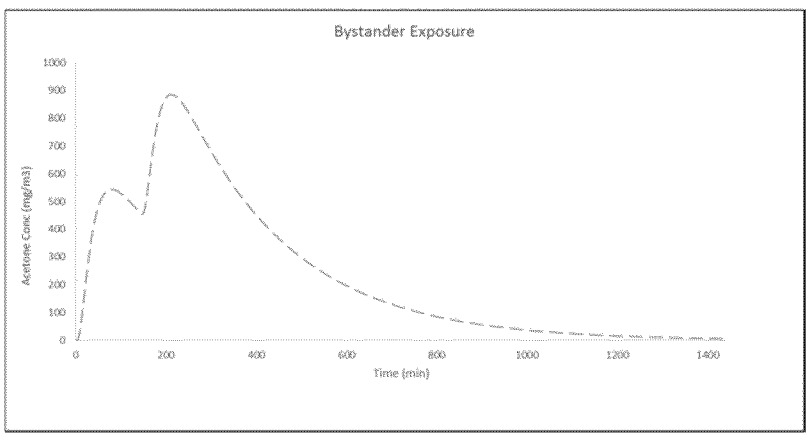


Acetone

Scenario #3: Brush-on application

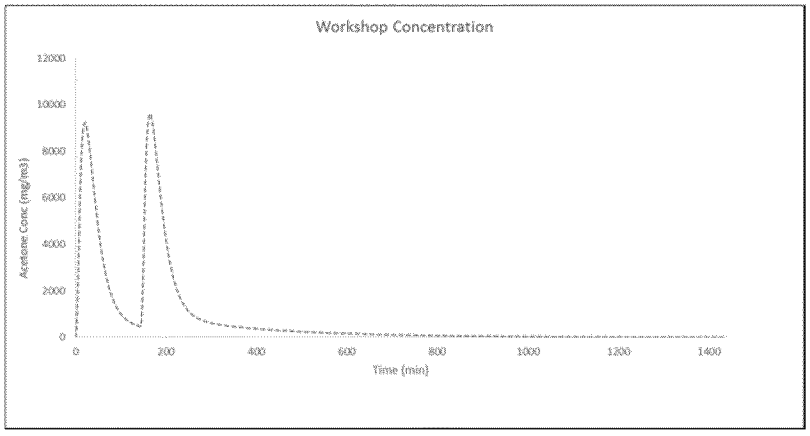


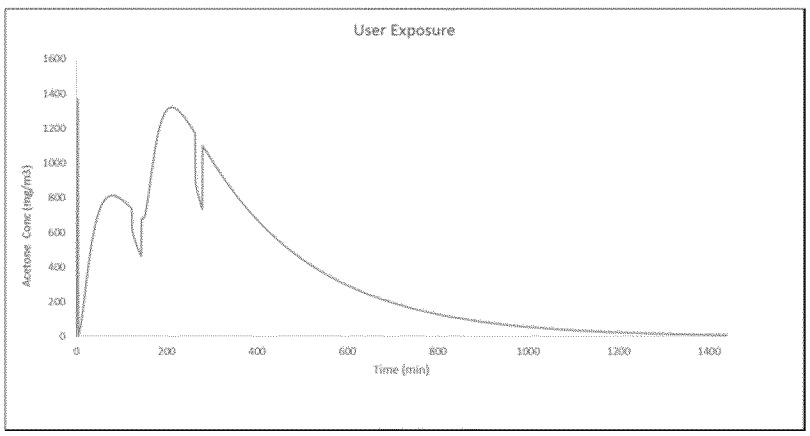


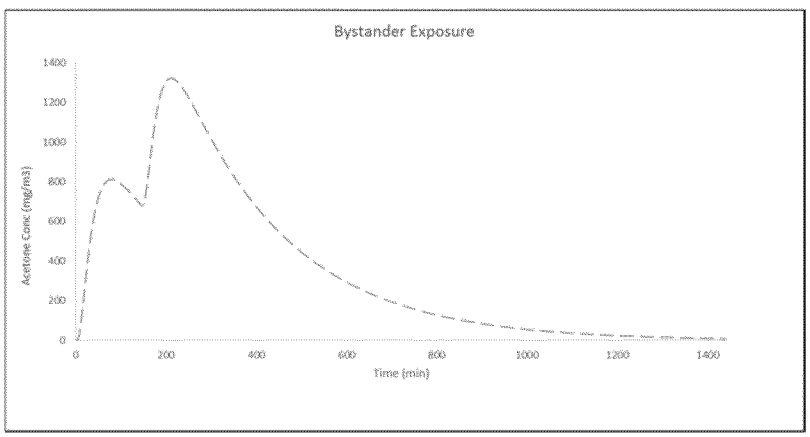


Acetone

Scenario #6: Spray-on application

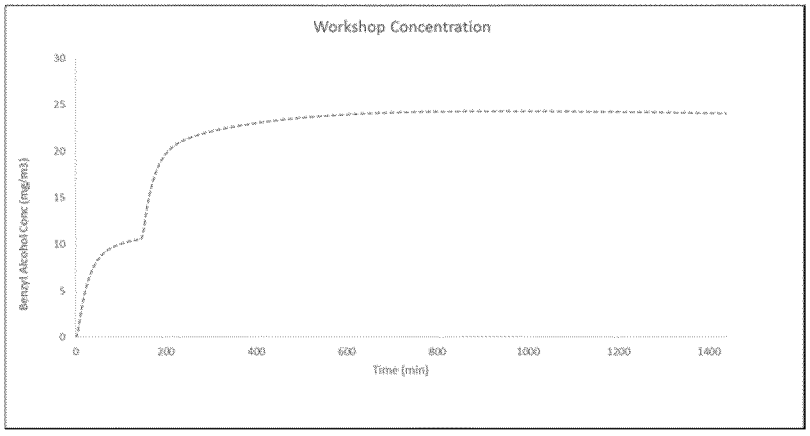


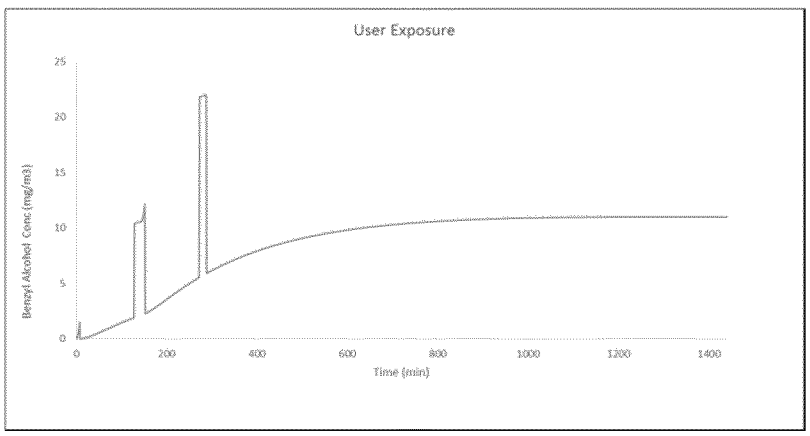


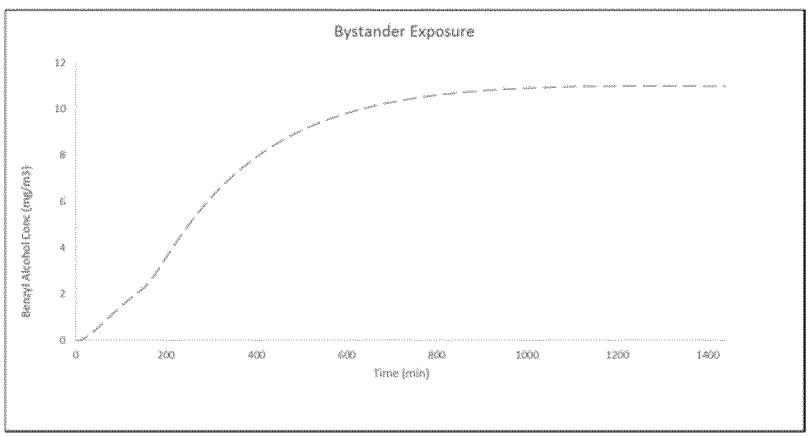


Benzyl Alcohol

Scenario #3: Brush-on application

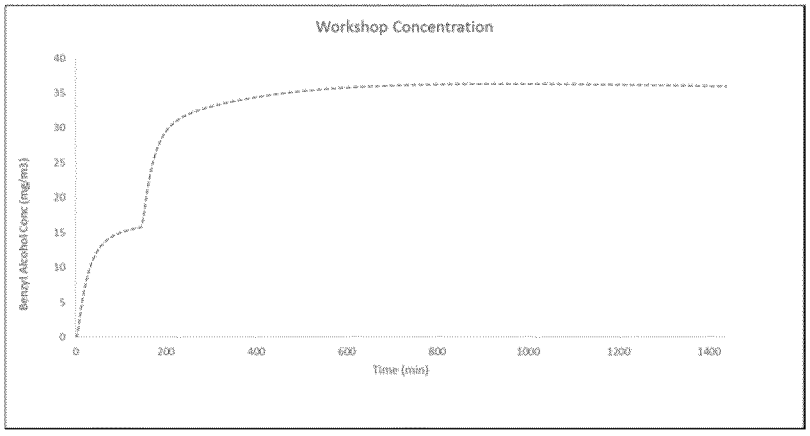


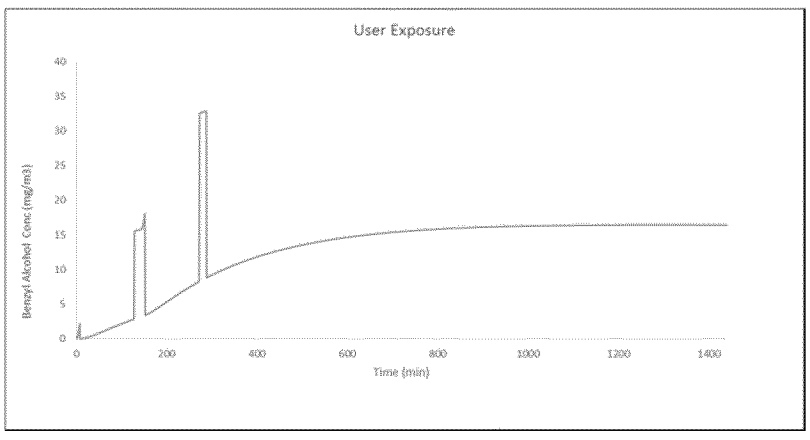


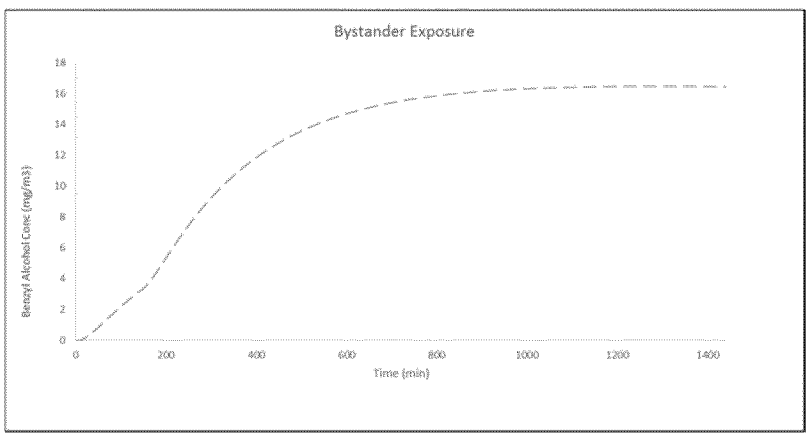


Benzyl Alcohol

Scenario #6: Spray-on application

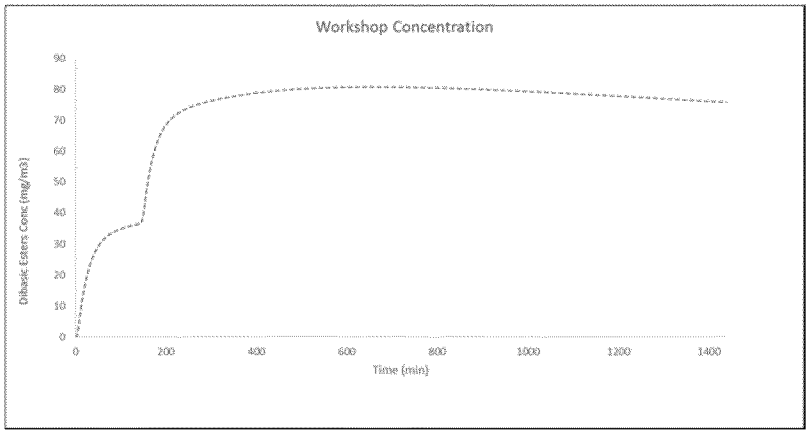


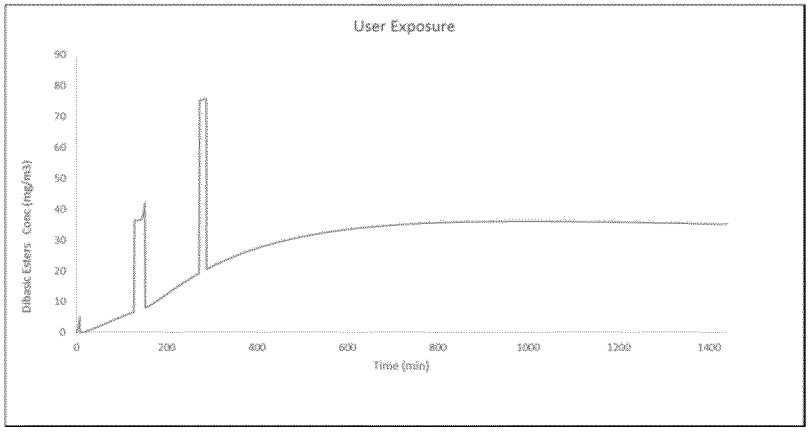


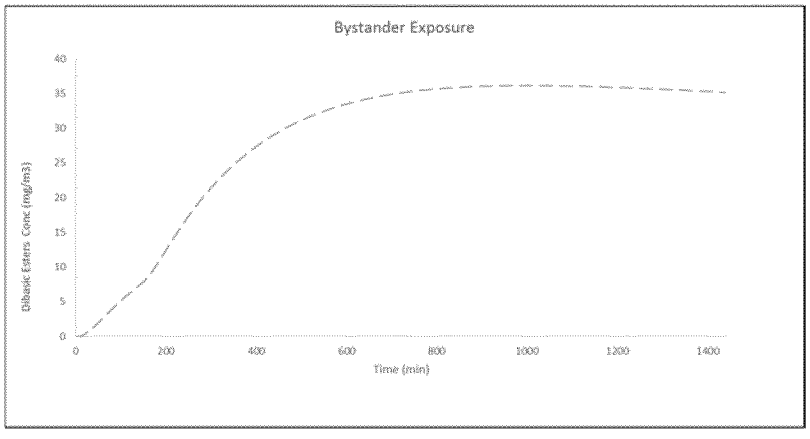


Dibasic Esters

Scenario #3: Brush-on application

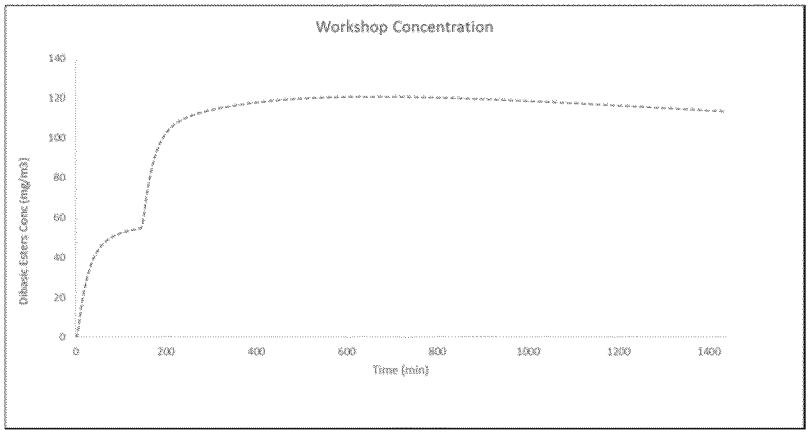


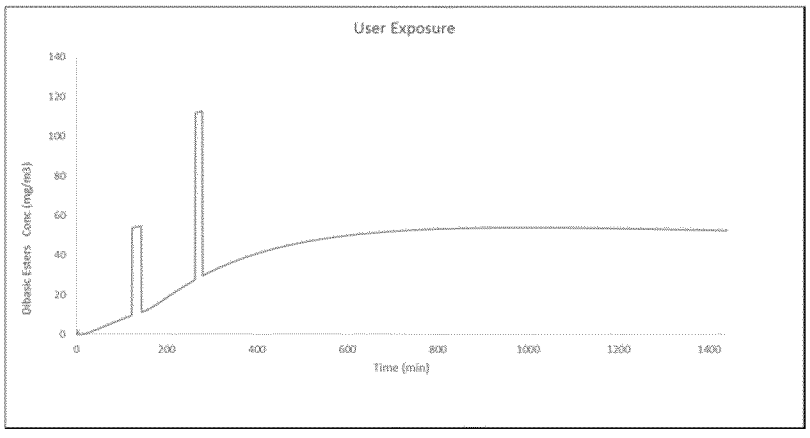


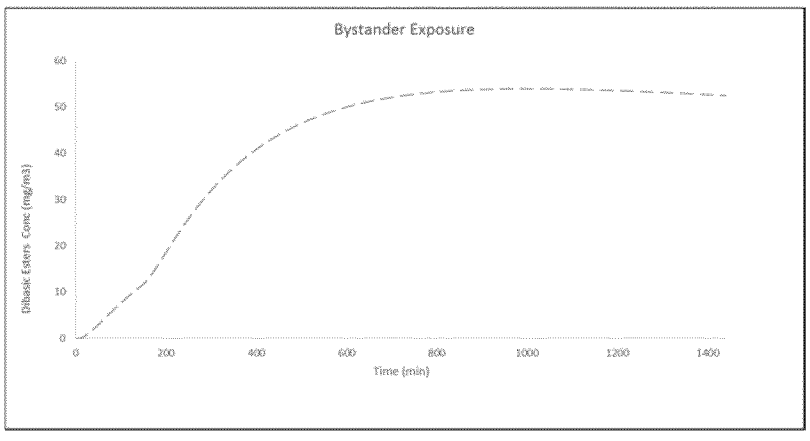


Dibasic Esters

Scenario #6: Spray-on application

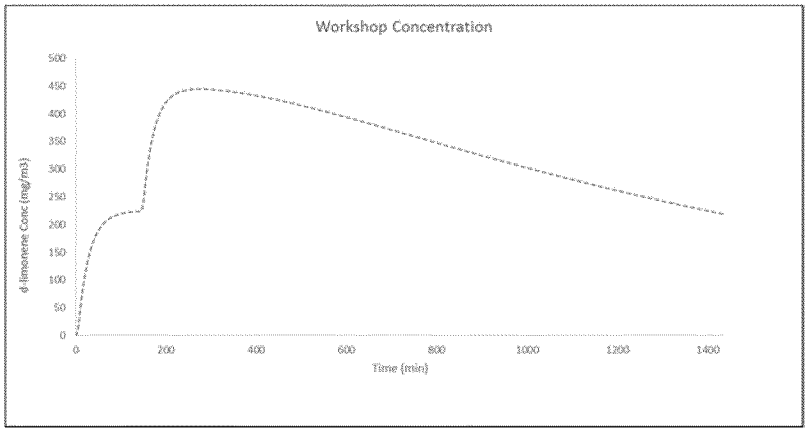


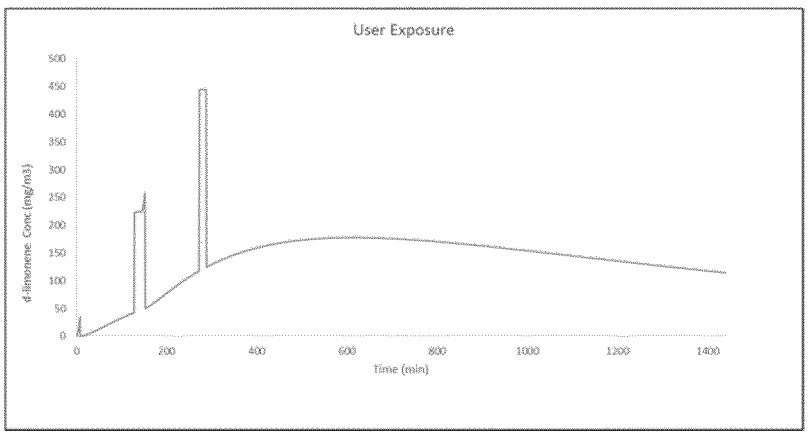


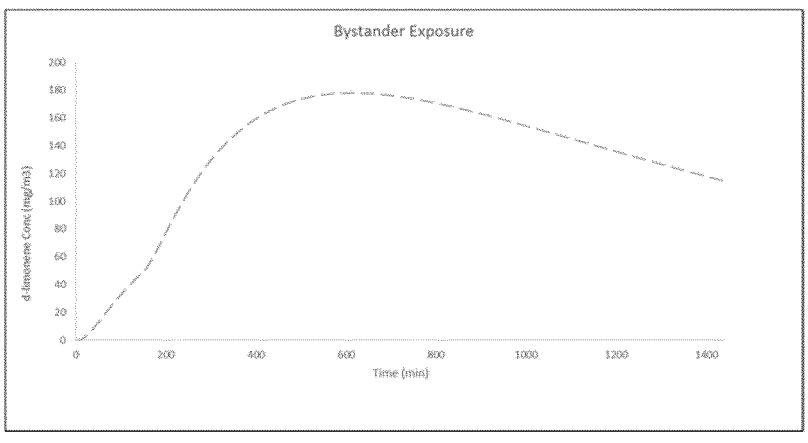


D-Limonene

Scenario #3: Brush-on application

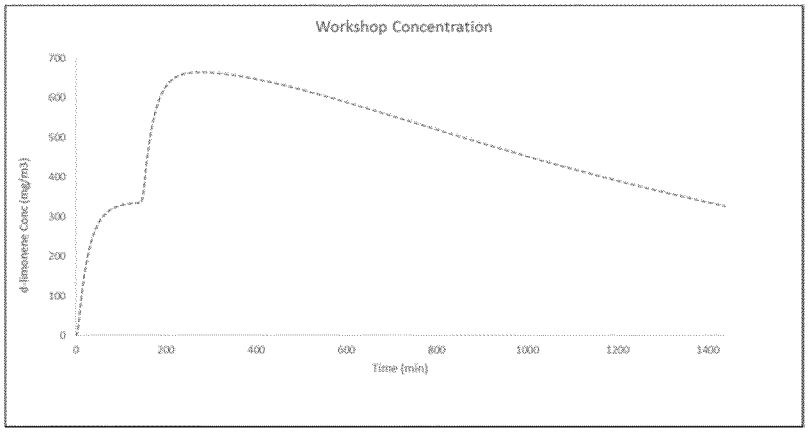


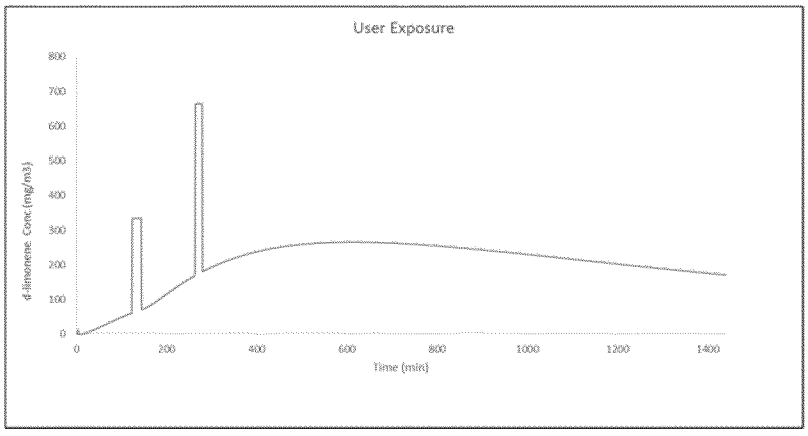


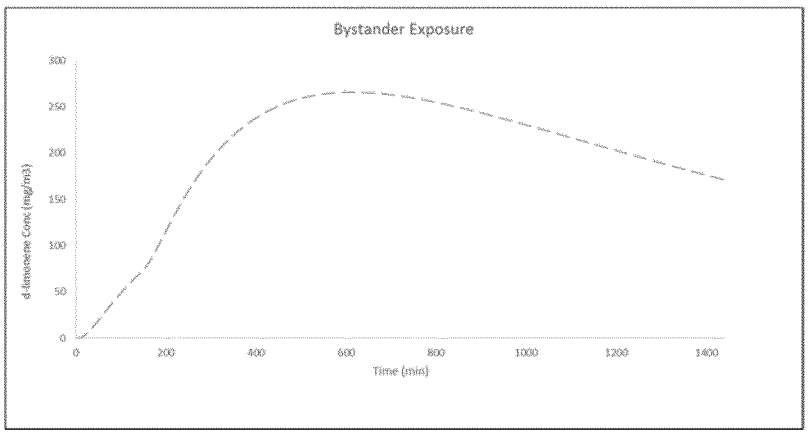


D-Limonene

Scenario #6: Spray-on application

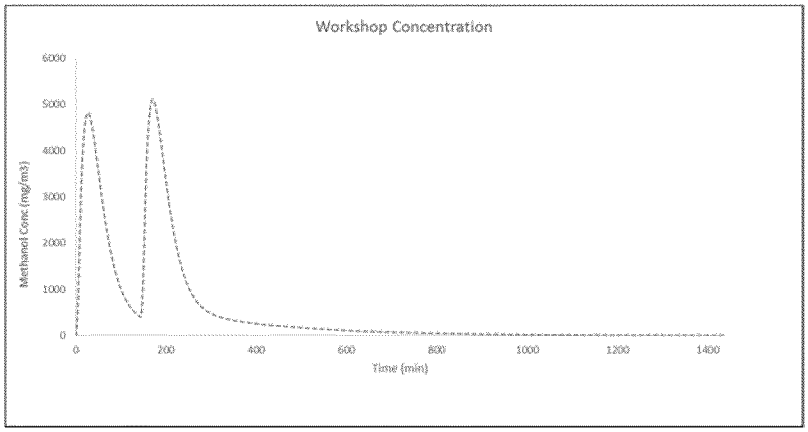


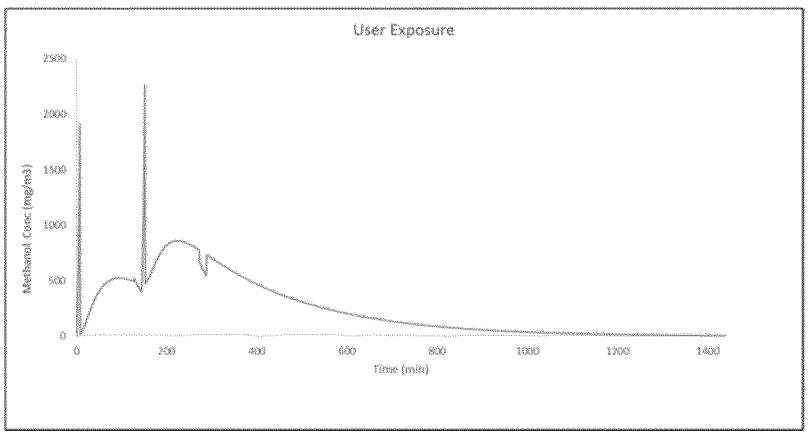


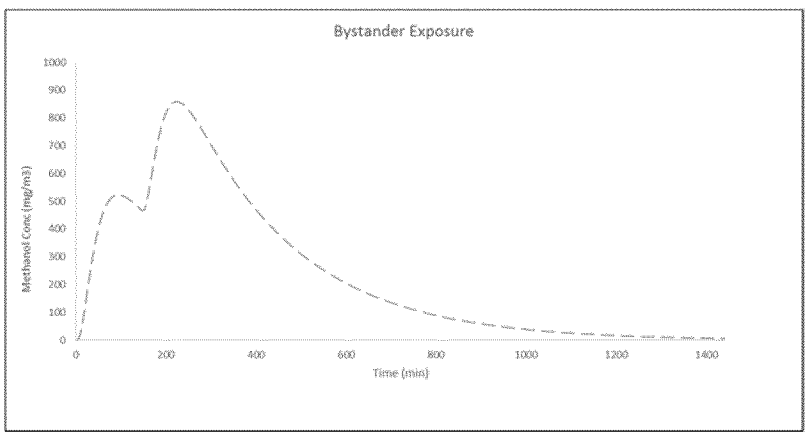


Methanol

Scenario #3: Brush-on application

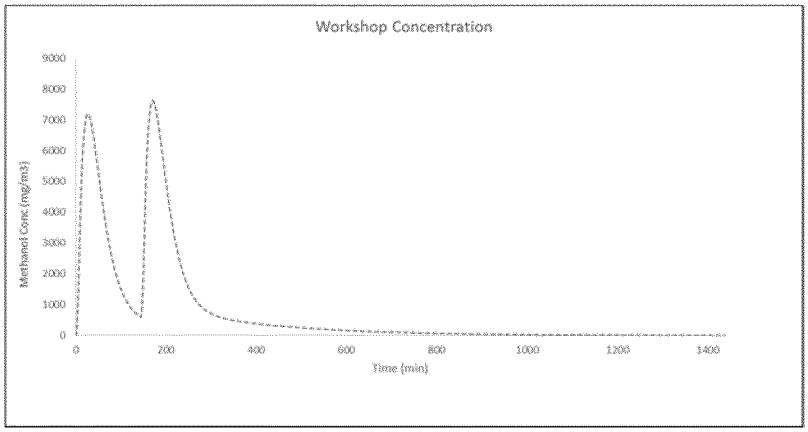


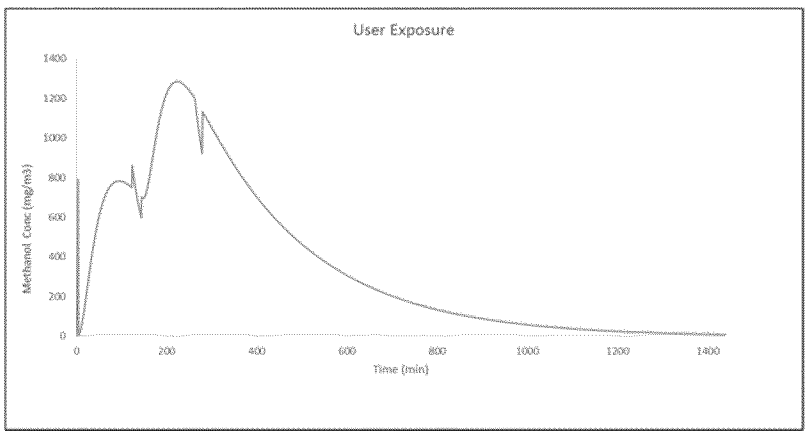


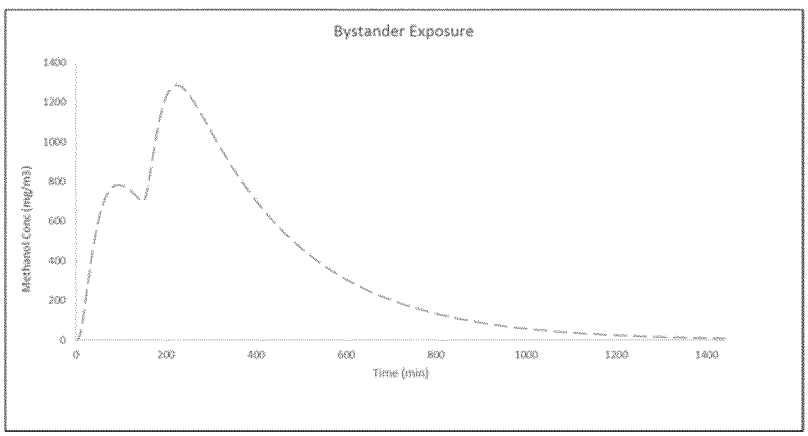


Methanol

Scenario #6: Spray-on application

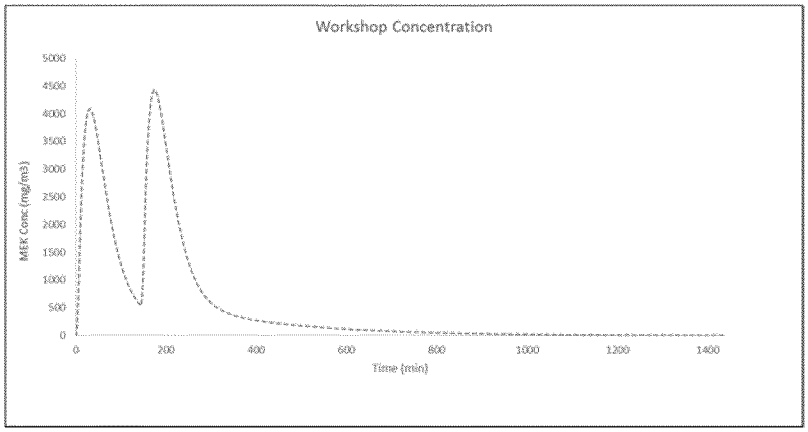


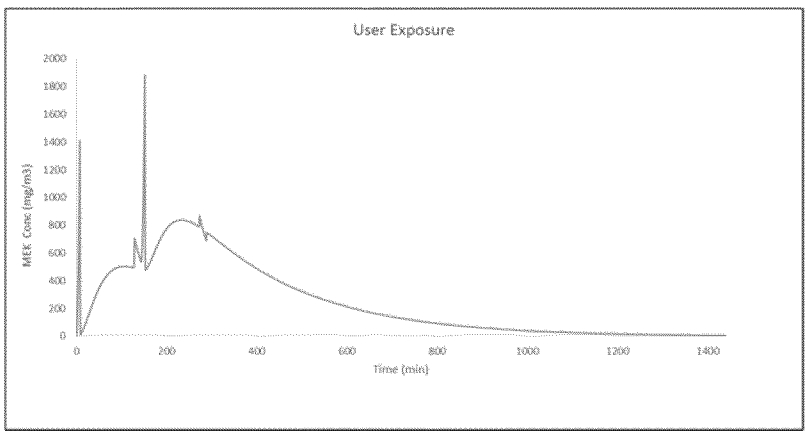


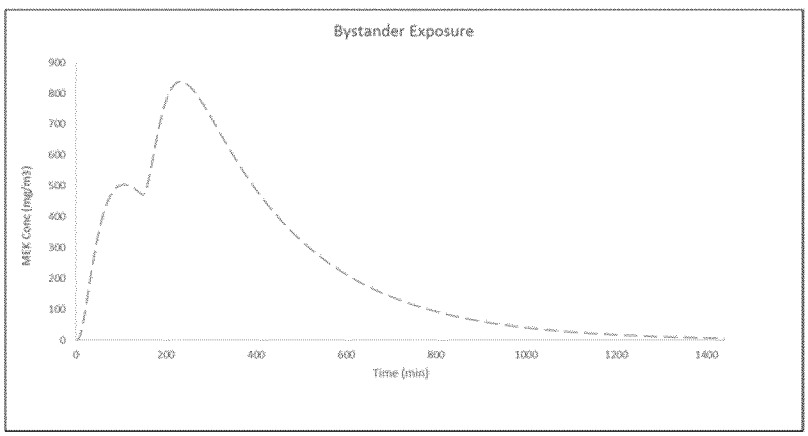


Methyl Ethyl Ketone (MEK)

Scenario #3: Brush-on application

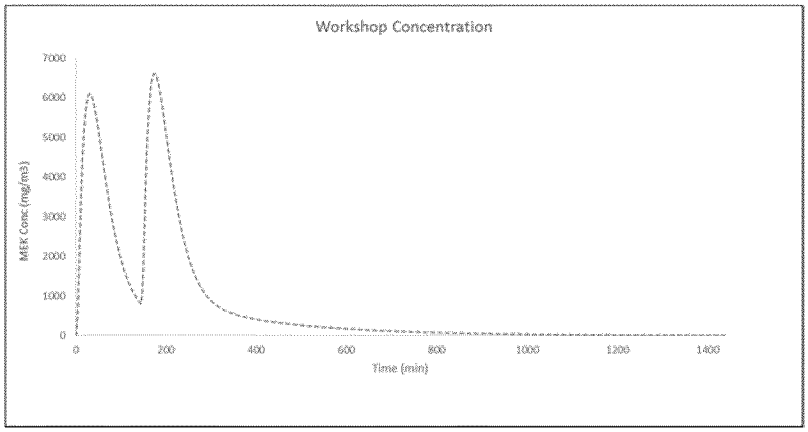


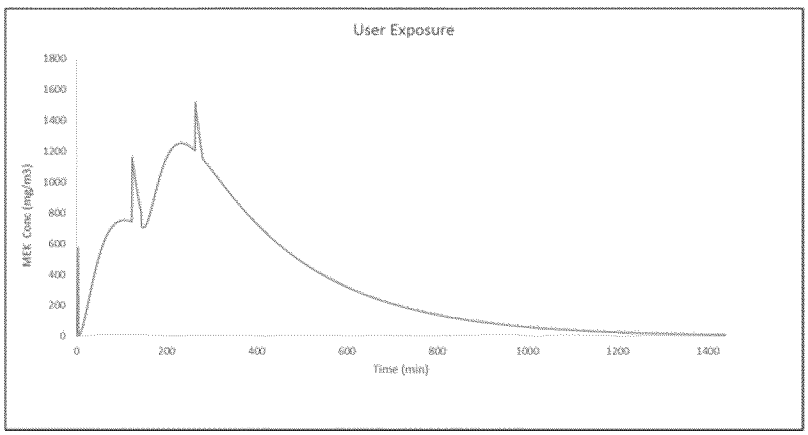


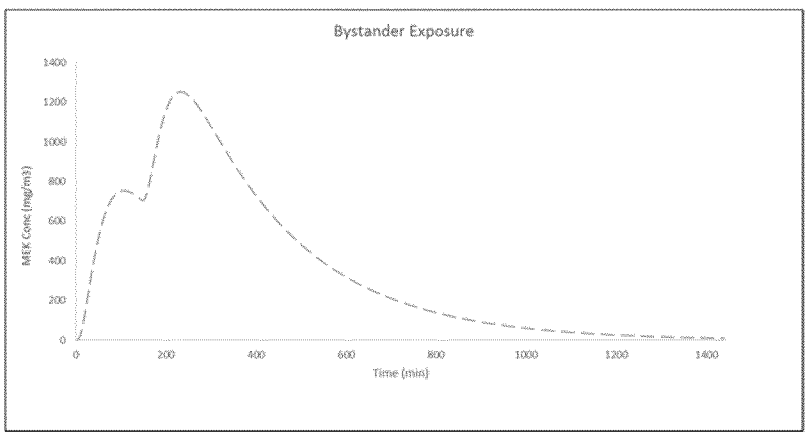


Methyl Ethyl Ketone (MEK)

Scenario #6: Spray-on application

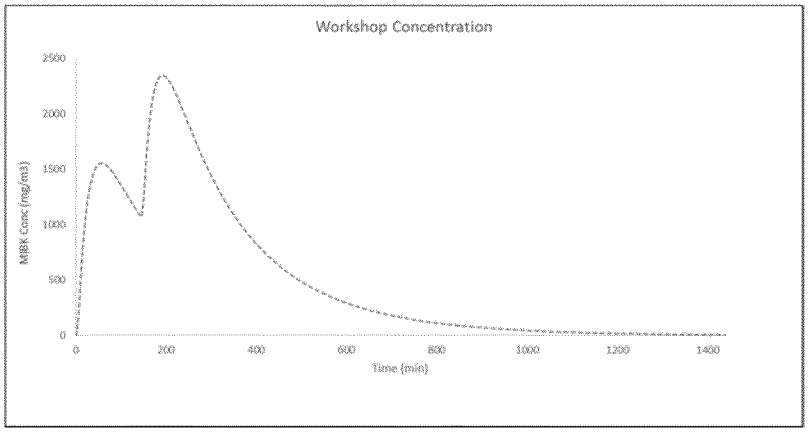


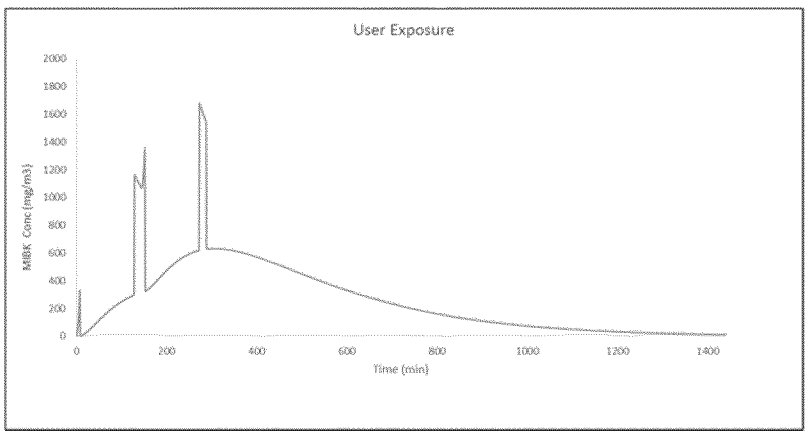


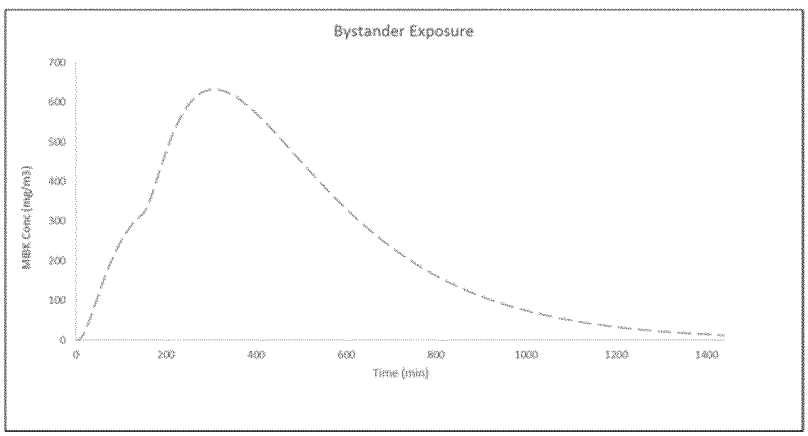


Methyl Isobutyl Ketone (MIBK)

Scenario #3: Brush-on application

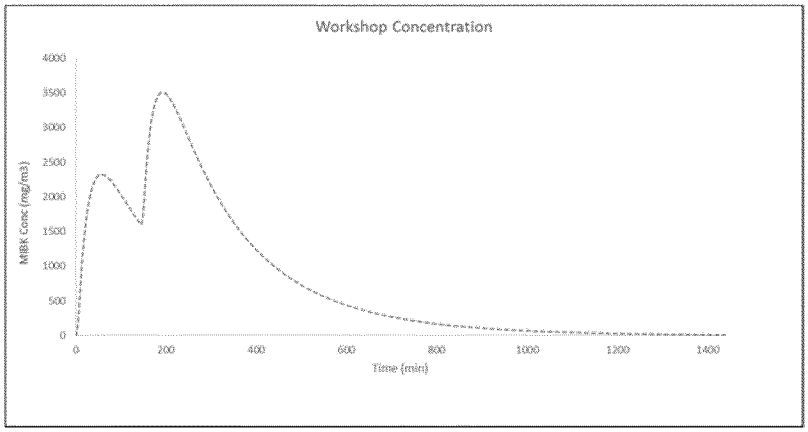


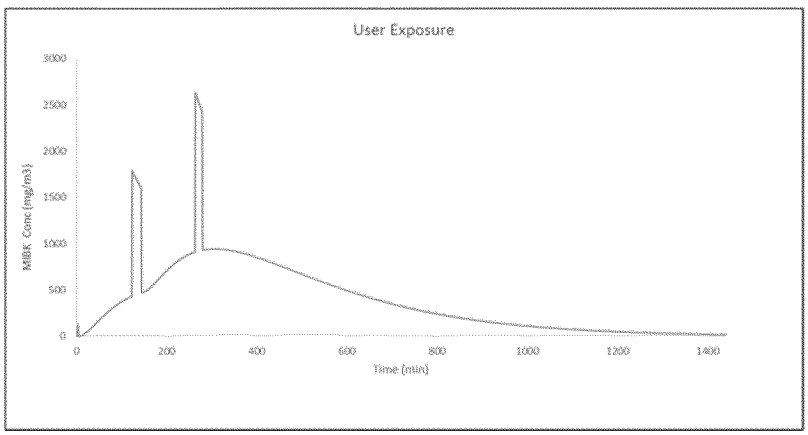


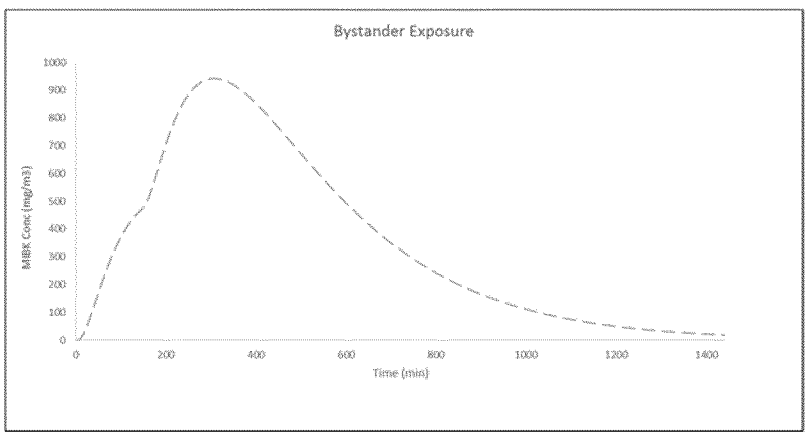


Methyl Isobutyl Ketone (MIBK)

Scenario #6: Spray-on application

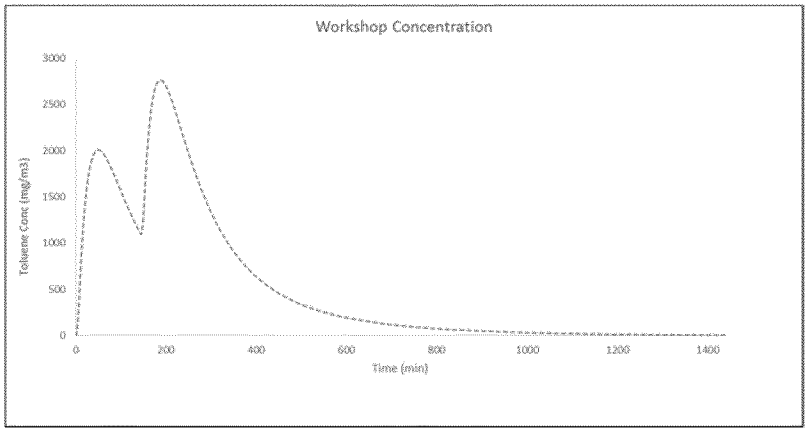


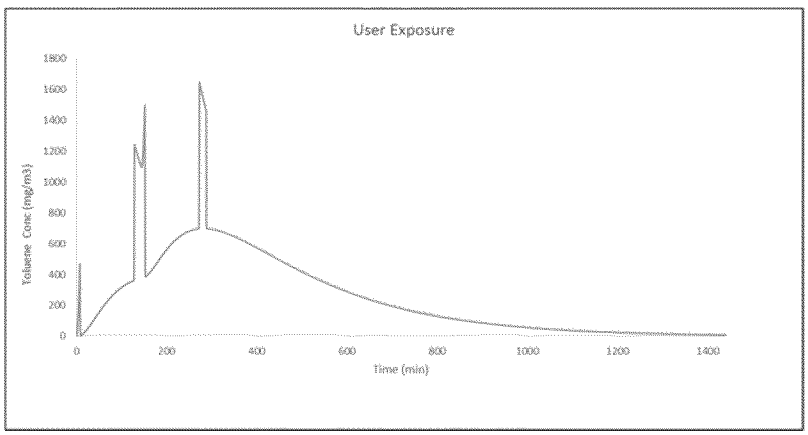


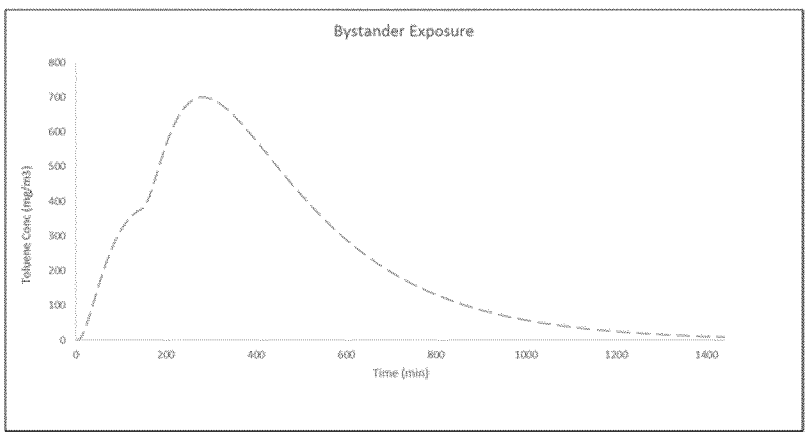


Toluene

Scenario #3: Brush-on application







Toluene

Scenario #6: Spray-on application